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SUBPERIOSTEAL ABSCESS IN THE FLOOR OF THE ORBIT ARNOLD KNAPP

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ARCHIVES OF OPHTHALMOLOGY.

RECURRENT PARALYSES OF THE EYE MUSCLES WITH ESPECIAL REFERENCE TO OPHTHALMOPLEGIC MIGRAINE.[1]

By Dr. A. DUANE, New York.

THE following cases are reported as illustrating unusual types of recurrent paralyses of the eye muscles.

Case 1.—Ophthalmoplegic Migraine; Recurrent Paresis of the Abducens. Milton S., aged 17. For two or three years has noticed that he sees double when looking to the right, but no deviation of the eyes was noticed until the present series of attacks.

For five months attacks of pain in the right eye, recurring first at intervals of two weeks and then lasting three or four days; afterward recurring at intervals of a week or less. At present, pain may recur at intervals of a few hours and then last but a few minutes. In severe attacks, pain associated with nausea and vomiting. No disturbance of vision during attacks, but latter are preceded by a period of irritability and depression.

For five months mother and other members of family have noticed that just before and during each attack patient looks decidedly cross-eyed. The strabismus usually lasts a week and then disappears. The attending physician says that in the intervals between the attacks there is no apparent deviation and no limitation of movement of the right eye outward.

[1] Read before Section on Ophthalmology, New York Academy of Medicine, April 16, 1923.

Status Præsens.—Movements of right eye outward less than normal by 3*mm:* movement of left eye outward slightly less than normal. Other movements normal. On tangent curtain homonymous diplopia, in right field only, increasing as eyes are carried to right. No vertical diplopia in any direction of the gaze. Pupils equal; light reaction sharp and equal in the two eyes; slight convergence reaction. Interior normal.

In this case there had evidently been for some time a slight right abducens paresis causing the diplopia that the patient had observed when the eyes were directed to the right, but no noticeable deviation. After this had lasted some years there developed attacks of right-sided migraine, conjoined apparently with marked exacerbation of the paresis. We say "apparently," for, while it is fairly certain that the periodic limitation of movement in the right eye and the accompanying characteristic diplopia were due to a weakness of the external rectus, the description is not sufficiently detailed to exclude absolutely a condition of periodically recurring convergence spasm.

Granting, however, that the condition is one of abducens paresis, it is noteworthy, first, as exhibiting one of the rather rare types of ophthalmoplegic migraine (see infra); second, because of the very short intervals between the attacks; and, third, because the exacerbations of paralysis apparently set in before the headache instead of afterward, as is the almost invariable rule.

Case 2. Recurring Incomplete and Partial Oculomotor Paralysis without Migraine.—Frederick H., aged 58. For twenty years has had at intervals of several years at least seven attacks of double vision unassociated with headache. Diplopia always vertical. During attacks there is no apparent deviation of the eyes. Present attack began two months ago. His physician reports that he has high blood pressure and albumin and casts in the urine.

Status Præsens.—In following a moving object shows left hyperphoria increasing moderately in looking up and to the right; diminishing as eyes are carried down, but well marked again in looking down and to the left. On tangent curtain diplopia increasing moderately in the same sense. Fields by rough test normal for large white objects. Interior normal.

Here there was a recurrent vertical diplopia, not very far from comitant, due, it would appear, to a moderate paresis of the right superior and left inferior rectus. The case is singular in that for twenty years the paralysis had remained incomplete and probably always confined to a single muscle in each eye, and that it was unassociated with migraine.

Still more aberrant were the following cases:

CASE 3. MIGRAINE ASSOCIATED WITH INTERMITTENT CONVERGENCE PARESIS.—Sophie K., aged 23. Quite anæmic. Had post partum hæmorrhage two and a half years ago and an operation afterward. Mother has migraine with scintillating scotoma.

For one and a half years almost constant bilateral supra-orbital pain. Photopsiæ frequent. Very marked asthenopia with pain in eyes. Objects within one or two feet look double.

Attacks in which there is first diplopia for five minutes, then a scintillating scotoma lasting ten to fifteen minutes, then severe pain with nausea and chilliness.

Status Præsens.—Vision normal, right and left; slight hyperopic astigmatism shown by test with homatropine. Exophoria 1–2▽ for distance, 8–10▽ (varying) for near. Near point of convergence at first 10 to 12*cm* from intercentral base line, but after a moment she shows crossed diplopia persisting when object of fixation is carried out to one meter. At another examination, diplopia persisted out to 5 meters, and patient was unable to make any effort to converge. In looking to either right or left the crossed diplopia is less than in looking straight ahead. Conjugate movements of eyes, and in particular the movement of each eye inward, normal.

Correction of the refraction ameliorated the headaches but produced little change in the muscular conditions.

Here there was a convergence insufficiency of only moderate degree, which very readily was converted into a convergence paralysis, producing insuperable crossed diplopia and rendering the patient temporarily unable to converge the eyes, although the power of inward rotation per se remained normal. This paroxysmal development of paresis and diplopia regularly preceded the attacks of migraine, which themselves were ushered in by the usual scintillating scotoma. This succession of symptoms, as before remarked, is very uncommon, for when

paralysis is associated with migraine it almost always follows the latter.

CASE 4. RECURRENT MIGRAINE WITH UNILATERAL MYDRIASIS.—Malcolm M. K., aged 20. Nine or ten months ago attack in which left pupil dilated and a scotoma developed, occupying the left half of each visual field. This was followed by very severe headache. Yesterday similar attack, only more severe. Left pupil, he says, was still somewhat dilated this morning. When later in the day he reported for examination, the pupils were equal and very mobile. Reactions to light (direct and consenual) and to convergence normal. Accommodation equal in the two eyes and normal. No limitation of movement.

After this had only occasional headaches.

Three years later examination on several occasions showed left pupil at times equal to the right, but usually under all conditions of testing 1*mm* wider. Both pupils wide (right 4–6*mm*, left 5–7*mm*) and very mobile. Reactions normal (contraction of left to convergence at least fully equal to that of right). Accommodation equal and normal in the two eyes.

In this case also the motor anomaly, which was confined to the iris, preceded the migraine. It remained as a more or less persistent condition three years later although the attacks of migraine had ceased or had become infrequent. The motor anomaly itself consisted either in a unilateral paresis of the sphincter, or, more likely, in a unilateral spasm of the dilatator iridis set up by sympathetic irritation.

TYPES OF RECURRING PARALYSES.

Recurring paralyses of the ocular muscles comprise two rather well-differentiated types, viz. those due to nuclear and those due to basal lesions.

Recurrent Nuclear Paralyses.—Recurrent nuclear paralysis is most frequently produced by tabes. Such paralyses are often quite transient and are frequently both partial and incomplete; they may be bilateral; and in recurring may affect a different muscle from that first involved. They may affect the exterior muscles, the interior being exempt, or vice versa.

According to Wilbrand and Saenger these recurring tabic

paralyses may be ascribed to a repeated toxic action of the syphilitic virus on the nuclei. When, as often happens, this repeated action produces degeneration of the nuclei, the paralysis becomes permanent.

Naturally, with paralyses of this sort, even in the transient stage, we find other evidences of tabes (Argyll-Robertson pupil, absence of patellar reflex, Romberg symptom).

While the great majority of tabic paralyses are nuclear, they may occasionally be due to a peripheral inflammation of the nerves themselves and so be of basal or possibly even of orbital origin (Dejerine and others cited by Wilbrand and Saenger).

Recurrent paralyses quite similar to those in tabes may be produced by diabetes, the lesion here consisting probably of minute hemorrhages into the nerve nuclei.

Recurrent nuclear paralyses may also occur in chronic progressive ophthalmoplegia. The characteristic marks of these cases are the absence of headache, the bilateral character of the paralysis, the peculiar fluctuations in its intensity, and the non-involvement of the interior muscles (Wilbrand and Saenger).

Other forms of recurrent paralysis due to nuclear lesion occur at times in chronic ophthalmoplegia combined with lesions of the bulbar nuclei and of the anterior horns and in bulbar paralysis (Wilbrand and Saenger).

Basal Recurrent Paralyses.—Basal recurrent paralyses may be due to a peripheral neuritis or to an intermittent compression of the nerves at the base resulting from the presence of a tumor, of vessels distended by aneurysms or other causes, or of inflammatory deposits. The cause of these lesions may be syphilis, tuberculosis, multiple neuritis, or trauma, and other cases have been ascribed to toxic conditions (autotoxis or focal infections), but in most cases the cause is quite uncertain. The type form of these paralyses is ophthalmoplegic migraine, which will be described more particularly below.

Some cases of recurrent paralysis may conceivably be orbital in site, being due to disease of the accessory nasal sinuses or to peripheral neuritis.

Cyclic Paralysis.—Mention must be made of the extremely rare condition called cyclic paralysis, cases of which have been described by Fuchs, Rampoldi, and Axenfeld and

Schürenberg. In this a condition of complete oculomotor paralysis with ptosis alternates every minute or two with rhythmic spasm of the levator and the interior muscles, so that the drooping lid rises, the eye converges slightly, the dilated pupil contracts, and the paresis of accommodation is replaced by a spasm, increasing the refraction by several dioptries. The condition is most common in females, and is either congenital or acquired very early in life. The site of the lesion is unknown.

OPHTHALMOPLEGIC MIGRAINE.

Historical.—As already stated the type form of recurrent basal paralyses is the condition which Charcot called ophthalmoplegic migraine.

The first mention of this condition seems to have been made by Notta (1854) who said that 6 out of 128 cases of trigeminal neuralgia showed ptosis and of these 6 three also showed an outward squint. Cases were published by Gubler in 1860, Mauthner (v. Graefe's case) in 1861, Saundby (1882), v. Hasner (1883), and Möbius (1884). The latter then defined the condition in terms that have been followed ever since. Afterward numerous other cases were published, and these have been summarized by various authors, e.g., Mauthner in 1889 (epitomizing 14 cases), d'Alché in 1896 (29 cases), Spicer and Ormerod in 1896 (30 cases), Mingazzini in 1897 (over 40 cases), Wilbrand and Saenger in 1900, Finlay in 1908, and Wilbrand and Saenger again in 1922. In all about 180 cases have been described, although it is likely that some of these were not genuine. For the descriptions are often very imperfect, and some of the summaries—especially the later ones—in which they are cited, contain not a few errors, the titles and the descriptions of the cases being sometimes quite inaccurate. I have sought, wherever I could, to verify the references given by Wilbrand and Saenger and others, but have by no means always succeeded. I have tried, however, in what follows to avoid inferences based on any description whose authenticity has not been verified or is not practically certain.

Characters.—As described by Möbius and most of the authors, ophthalmoplegic migraine is a condition, developing

in childhood or infancy, and characterized by irregularly recurring, severe, usually one-sided headaches, which are associated with nausea and vomiting but not with scintillating scotoma, and which after lasting for days or even weeks disappear and are replaced by a unilateral oculomotor paralysis usually on the same side as the head pain. This paralysis, which may develop before the migraine subsides completely, is at first partial and incomplete, but soon becomes total, *i.e.*, affects all the muscles supplied by the nerve, including the interior muscles and the levator, and also becomes complete. After a variable time—days or weeks—the paralysis subsides only to recur at some time later in conjunction with another attack of head pain. This regular association of paralysis with migraine-like attacks may occur at the very outset of the trouble, but in most cases there is a pre-paralytic period during which the patient suffers simply from the recurring head pains; then after these have been going on for a long time—it may be many years—the paralytic attacks set in.

In the intervals between the paralytic attacks the paralysis may disappear completely (intermitting paralysis) or may remain in part (remitting paralysis). As the disease goes on the tendency of the paralysis to persist in the intervals becomes more marked, and finally—frequently after the lapse of a number of years—it does not disappear at all, so that one oculomotor nerve remains permanently and completely paralyzed. The migraine attacks keep on whether the paralysis intermits, remits, or becomes permanent.

At first it was thought that the condition was more frequent in females, but in the cases so far recorded in which the sex was noted 77 were males and 66 females.

Variations in Symptoms and Course.—Such is the type form of ophthalmoplegic migraine. Some would have it that it is the only form, *i.e.*, that the disease always begins early in life, always affects the oculomotor nerve and no other, is always complete and total, and always recurs in the same eye. But these statements are too sweeping, for there are many undoubted cases of the condition which do not conform to them.

1. The paralytic attacks do not always begin in early life. It is true that in the great majority of cases they begin before the age of 20, and the migraine attacks begin earlier still—

usually in childhood. In some cases, indeed, the symptoms develop very early, paralysis setting in at the age of 8 months (Shaw), 11 months (Möbius), one year (Case 2 of Spicer and Ormerod, Case 2 of De Schweinitz), one and a half years (De Schweinitz' Case 1, Snell, Wolff, Leavitt), two years (Koch, Rozsa, Steenhuisen, Case 3 of Spicer and Ormerod), or three (Klinedinst). On the other hand, in two of Wilbrand and Saenger's cases the paralysis set in at the ages of 25 and 33, in three of Fisher's at 30, 32 and 33, in Case 1 of Danis's at 27, in Case 1 of Charcot's at 35, in Strzeminski's at 37, in Dabney's at 45, in Chabbert's at 52 and 53, and in Darquier's at 63.

2. The paralysis may develop before the patient begins to have migraine attacks at all, as in cases reported by Hinde and Moyer, by Shaw, and in my Case 1.

3. Migraine may be absent altogether, the patient having recurrent paralytic attacks, otherwise characteristic. Thus there were recurrent attacks of oculomotor paralysis without migraine in my Case 2, in the cases reported by Dabney (followed for 17 years), Gubler, Weiss, Valude and Frogé, and in two cases reported by Parenteau; and a recurring abducens paralysis without migraine is reported by Spicer and Ormerod (Case 5).

4. In very rare cases in the attack the paralysis may precede the migraine. This apparently happened in my Case 1, as well as the aberrant Cases 3 and 4, and in the cases of Snell, in which the attack was ushered in by ptosis. In Colman's case, a sort of motor spasm preceded the migraine; *i.e.*, the upper lid flapped up and down at intervals during a quarter of an hour, then migraine set in followed by third-nerve paralysis with ptosis. This curious flapping motion of the lid was repeated during succeeding attacks, even after the third nerve paralysis including the ptosis had become permanent.

5. Not infrequently after the patient has begun to suffer from attacks of paralysis with migraine, these attacks alternate with attacks of simple migraine without paralysis. This was noted in at least 14 cases and may very likely have occurred in a number of others, whose histories are imperfectly recorded. It seems also that it is possible to abort an attack so that it does not go on to paralysis. A patient observed by Alger, who

suffered from typical ophthalmoplegic migraine, asserted that he could cut off an attack by going to sleep or indulging in sexual intercourse.

6. The head pain is occasionally preceded by a scintillating scotoma. This was present in my two aberrant cases (Cases 3 and 4), and in cases reported by Chabbert, Spiller and Posey, Romano, Fisher (Cases 4 and 5), and Wilbrand and Saenger (Case 2).

7. The paralysis during an attack is by no means always either complete or total. Sometimes it is partial and incomplete during the first attack and complete and total later; at times it remains partial throughout the disease or, at least, during the period of observation. This was seen in my Case 2 and in quite a large number of other cases reported.

Of special interest are the cases in which the recurring paralysis is confined to the interior muscles. Apart from Hansell's cases of recurrent ophthalmoplegia interior, of which probably perhaps only one belongs in the condition here under consideration, and apart from my somewhat dubious Case 4, Troemner reports a cure of recurrent migraine with unilateral ophthalmoplegia interior, and Wilbrand and Saenger report (Case 3) a very interesting case of ophthalmoplegia interior with migraine recurring with menstruation. I saw in the care of another physician, who will report it, a case of recurrent complete paralysis of accommodation with migraine occurring in a young girl.

8. The paralysis may affect other nerves than the oculomotor. Isolated recurring paralysis of the abducens is not so very rare, occurring in about 8 per cent. of the cases of ophthalmoplegic migraine. Thus it was present in my Case 1 and in cases reported by De Schweinitz (Case 2), Wolff, Spicer and Ormerod (Cases 6 and 7), Fisher (Cases 4 and 5), Seele, Armaignac, Bernheimer, Brav, Leavitt, Veasey, and Danis.[1] With regard to these cases I would point out the possibility, already touched on, of an error in the diagnosis, due to the fact that a paralysis of the abducens may be simulated by a convergence spasm when the latter is of such intensity as to prevent the movement of either eye outward. This cause of

[1] In Marina's case, often referred to as one of simple abducens paralysis, the 3d and 6th nerves were both affected. (See below).

error can usually be eliminated if we cover the non-deviating eye, thereby breaking up the strong tendency to converge. Then, if the case is one of convergence spasm, the eye can be got to move well out, whereas it will not do so if the case is one of abducens paralysis.

9. So also, but more rarely there may be a typically recurring isolated paralysis of one superior oblique. Such cases have been reported by Fisher (Case 2),[1] Bornstein, v. Luzenberger, and Scremini (this case and Fisher's, however, had but one attack).

10. Isolated recurrent paralysis of the facial, associated with migraine, also occurs (Rossolino, Hatschek). Bernhardt reports four unilateral cases, some with, some without pain; but it is doubtful if any of them belongs to the type of paralysis here considered. In one of these cases, there was apparently recurrent paralysis of the auditory nerve as well.

11. There may be paralysis of several nerves on the same side. The combinations reported are:

(a) Paralysis of oculomotor and abducens. Cases reported by Charcot (Case 2), Carpenter, Contouzis,[2] Mendel and Marina. In Carpenter's case there was first a simple oculomotor paralysis, then an abducens paralysis was added.

(b) Paralysis of oculomotor (exterior branches), abducens, and hypoglossus (Sil). Compare also Fisher's Case 4 in which at one time the hypoglossus, at another the abducens was affected.

(c) Paralysis of the oculomotor and trigeminus (Haynes, Vissering). Combinations of oculomotor paralysis with hyperæsthesia, anæsthesia, and paræsthesia in areas supplied by the trigeminus are fairly common.

(d) Paralysis of oculomotor, abducens, and trochlear nerves, *i.e.*, complete unilateral ophthalmoplegia (Brissaud, Sciamanna). Carpenter's case seems ultimately to have become a nearly complete total ophthalmoplegia. Gasparini's was

[1] Fisher's Case 3 may also have to be included here, although the description of the diplopia (diplopia in the sense of a right hyperphoria increasing to the left) leaves one in doubt whether the case was one of paralysis of the left superior rectus or the right superior oblique.

[2] Contouzis says paralysis of third and fourth pairs; but from his description it is evident that this is a slip of the pen.

one of unilateral ophthalmoplegia exterior. (Parenteau's case stated to be an ophthalmoplegia was a 3d nerve paralysis).

(e) Paralysis of abducens and later of facial also (Spicer and Ormerod's Case 5).

(f) Paralysis of abducens and superior oblique of one eye with diplopia indicating possibly a paresis of the superior rectus on the same side or more likely a paresis of the superior oblique on the other (Paderstein's Case 2).

(g) Paralysis of the abducens superadded to a divergence paralysis (Winawer).

(h) A rather uncommon combination is that of paralysis of the eye muscles with disturbances of the auditory nerves, evidenced by tinnitus or deafness.

(i) Motor paralysis associated with affections of the optic nerve and also with visual disturbances without evident cause, sometimes occur. Thus, apart from some cases in which there was congestion or inflammation of the nerve head, others occasionally occur in which there is amaurosis or amblyopia of the affected eye without apparent lesion.

In Spiller and Posey's case the blindness which lasted half an hour and was accompanied by photopsiæ seems to have been simply an exaggerated form of the scintillating scotoma which precedes a migraine. But in Fisher's Case 4 the blindness did not wholly disappear for two or three days and in the case described by Kollarits it lasted three weeks and left a permanent amblyopia. In five other cases cited by Wilbrand and Saenger, in Rozsa's case, and in one (Saundby's) cited by Spicer and Ormerod the motor paralysis was associated with either amblyopia or amaurosis. It is questionable, however, in some of these cases whether the amblyopia was not a pre-existent condition and thus independent of the ophthalmoplegic migraine.

In one or two cases the visual field has been contracted. Such a contraction has been thought to indicate a functional disturbance (hysteria), but in the case described by Thomsen and Richter a contraction of the field increasing and diminishing with the paralysis was associated with an organic lesion of the oculomotor nerve.

(j) The paralysis may be associated with paræsthesiæ of various kinds, disturbances of secretion, fever, convulsions, etc.

12. Sometimes the paralysis is bilateral. The following combinations are reported, some of which, however, quite certainly do not belong with true ophthalmoplegic migraine.

(a) Paralysis of a single muscle in each eye (my Case 2).

(b) Oculomotor paralysis alternating between the two eyes[1] (Darquier).

(c) A gradually developing oculomotor paralysis, at first alternating between the two eyes, but ultimately terminating in an almost complete bilateral ophthalmoplegia exterior (Spicer and Ormerod's Case 4). Somewhat similar was the case of Chabbert.

(d) Ophthalmoplegia exterior alternating between the two eyes (Ziehen).

(f) Oculomotor paralysis in one eye and abducens paralysis in the other (Charcot).

(e) Ocular paralysis recurring every year for 24 years, ending finally in total bilateral ophthalmoplegia, respiratory paralysis, and death (Duboys).

(g) Bilateral total oculomotor paralysis combined with bilateral, nearly total paralysis of the abducens and probably also paralysis of the trigeminus (Leclerc).

(h) Oculomotor paralysis in one eye and facial paralysis in the other (Saundby).

(i) Bilateral oculomotor paralysis combined with facial paralysis (Cantalamessa).

(j) Alternating oculomotor paralysis with at times paralysis of the facial or of the facial and abducens on one side (Pflüger).

(k) Alternating paralysis in which the left facial, the left abducens and hypoglossus, the right facial and auditory nerves, the right abducens and left facial, and the right facial were affected in successive attacks (Nieden).

(l) Alternating paralysis of facial alone (case of Rossolino and one of Bernhardt's).

(13) The course is not always that described. While in most cases the paralysis tends more and more to persist in the intervals between the attacks and ultimately becomes permanent, there are cases in which, at least for many years, it remains

[1] Wilbrand and Saenger (I. p. 493) cite Anderson's case reported by Jack, as a bilateral case (ptosis in both eyes). But as Jack points out, the ptosis in the left eye was apparent only.

intermittent. Thus it so remained for 20 years in my Case 2, for 13 years in the case reported by Danis, 14 in Spicer and Ormerod's Case 1, and 17 in Dabney's case. (The last case is particularly convincing since Dabney had it under observation during the whole time). In Karplus's third case, the paralysis had recurred for over 42 years without becoming total. In other cases the attacks, instead of increasing, even lessen in severity (cases of Parinaud, Priestly Smith). This was noted as a temporary happening in some of the progressive cases. Finally, there are a few cases in which there is a cure or, at least, a period of many years in which attacks are absent (cases of Clarke, Morgano, Bernhardt's Case 1). Bernhardt's case is specially remarkable since the disease had begun some forty years before.

Nature of Ophthalmoplegic Migraine.—The symptoms of typical ophthalmoplegic migraine make it certain that the condition cannot be nuclear in origin. For most of the cases exhibit sooner or later a total and complete paralysis of the third nerve on one side, and for such a paralysis to be nuclear would require lesions distributed in a quite peculiar fashion in both oculomotor nuclei. The condition must, in fact, be due to injury of the radicles or trunk of the oculomotor nerve itself, in the latter case being basal.

This view is supported by the results of pathological investigation. The cases that have come to autopsy[1] show either a benign tumor, an inflammatory exudate, or a tuberculous mass involving the nerve and producing degeneration of its fibers. It seems likely, indeed, that a basal organic lesion of some sort is the cause of most of the typical cases of ophthalmoplegic migraine. Periodic variations in the size of a compressing growth or periodic variations in the size of the adjoining organs (blood-vessels, etc.), would account for the attacks and the remissions, and permanent paralysis would set in when the pressure had produced destructive changes in the nerve.[2]

[1] Reported by Gubler, Weiss, Karplus (2d case), Richter-Thomsen, Shinoya. In Duboys's case, which does not probably belong here, there was found simply meningeal congestion. No microscopic examination, however, was made.

[2] This conception of the condition is practically that enunciated by P. C. Knapp is his survey of the condition many years ago.

Shinoya thinks that the reason why the oculomotor nerve is predominantly affected is that the latter lies between the posterior cerebral and the superior cerebellar arteries. Von Luzenberger thought that the recurrent trochlear paralysis in his case was due to periodic distention of the cavernous sinus producing intermittent pressure on the first branch of the trigeminus and on the trochlear nerve.

There seems no reason why recurrent isolated paralyses of the fourth and sixth nerves with head pain should not be accounted for in the same way as similar isolated paralyses of the third nerve. They are doubtless likewise due to basal lesions and should be classed with ophthalmoplegic migraine. The like is probably true of most of the cases of combined paralyses, *i.e.*, paralyses of two or more nerves on the same side. More dubious are the cases of recurrent bilateral paralysis, especially those which exhibit the type of an ophthalmoplegia exterior or in which the successive recurrences differ in character. Such paralyses are more likely to be nuclear. The same is true of bilateral picked paralyses, like my Case 2.

Wilbrand and Saenger would differentiate from true ophthalmoplegic migraine the very similar recurrent paralyses with head pain caused by chronic meningitis, multiple neuritis, or internal hydrocephalus. But if these processes by intermittent pressure on the individual motor nerves at the base produce the symptoms of an ophthalmoplegic migraine, it seems difficult to see why this name should not apply to the paralysis so called, just as it does to paralyses caused by a basal tumor.

On the other hand, the condition should be differentiated from the paralyses of nuclear origin, especially the ordinary tabic paralyses or from a progressive ophthalmoplegia exterior. Yet, just as in rare cases some tabic paralyses are of basal origin, so some of the more aberrant forms of ophthalmoplegic migraine may be nuclear. My Case 2 of recurrent bilateral paresis without migraine was doubtless nuclear, and my Case 3 of recurrent convergence paralysis with migraine must have been nuclear or supranuclear.

The relation between the ophthalmoplegia and the migraine has been much disputed. Charcot, followed still by many authorities, believed that the migraine was the cause of

the paralysis in the sense that the recurrent vaso-motor disturbance produced by the migraine finally induced or rather favored the development of structural changes.[1] Others hold the reverse view, *i.e.*, that a structural condition exists which by causing pressure alike on the trigeminus and the motor nerves sets up both the migraine and the paralysis. The second hypothesis seems to explain better the totality of symptoms of this protean condition,[2] and is not incompatible with the fact that the migraine may exist for years before paralysis develops.

Möbius and others have objected to the term migraine as applied to the paroxysmal headache that accompanies the disease. They say that this differs from a true migraine in the absence of a family history of migraine, the absence of a scintillating scotoma, the long duration of the headache as well as the long duration of the intervals, and the fact that the head pain begins in childhood or infancy instead of after the age of puberty and tends to persist steadily through life instead of finally disappearing with age. To this it may be answered that a familial character is not so infrequent in this disease, that a scintillating scotoma is sometimes present, and that the head pains present many of the characters of a true migraine, *e.g.*, its unilateral character and its association with nausea, vomiting, and physical depression. Furthermore, migraine itself is more or less protean in its manifestations, so that we

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[1] It seems worth while to quote the precise words of this great clinician. After citing Gubler's and Richter's autopsies, the former showing a tuberculous growth, the latter a benign neoplasm, he says: "Il est clair que les productions bacillaires dans un des cas, le fibro-chondrome dans l'autre, n'ont pas été les agents primitifs et uniques de l'évolution du processus morbide. La périodicité des accès, la complete disparition des accidents dans leurs intervalles, du moins à l'origine, l'ancienneté du mal enfin remontant à l'enfance ne permettent guère de l'admettre. C'est seulement par la repetition multipliée des crises que les lesions, purement dynamiques et essentiellement temporaires d'abord, ont laisé subsister après elles une épine et un point d'appel, un lieu 'de moindre resistance,' sur lequel se sont fixés, de preference, sous l'influence de l'etat diathésique et independamment de l'affection migraineuse, les produits néoplasiques."

[2] Especially the cases in which migraine is absent and those in which paralysis develops with the very first attacks of migraine or in which it actually develops before the latter.

should not say that a head pain is not a migraine, simply because it lacks some of the ordinary characters of the latter.

As direct exciting causes of the condition have been given traumatism (v. Graefe's case reported by Mauthner, Richter-Thomsen, Platenga, Gubler); uric-acid poisoning (Priestley Smith); autoxis from intestinal putrefaction (several authors on what seem uncertain grounds). Other cases were associated with disease of the accessory sinuses, otorrhœa, and bad teeth, but the causal relation between these conditions and the paralysis is not clearly proved. Cases have been reported in which the paralysis recurred with attacks of typhoid or typhus fever and with successive attacks of relapsing fever, as in Mironesco's patient. Other cases have been ascribed to malaria. In several it has been noted that attacks recurred with menstruation (cases of v. Hasner, Morgano, and Romano and Wilbrand and Saenger's Case 3, in which there was a recurrent ophthalmoplegia interior); and Kipp, Mariana, and Brav report cases in which paralysis recurred in successive pregnancies.

A few cases have been regarded as purely functional (hysterical), but the evidence for this does not seem very convincing. As before noted, the varying contraction of the visual field occasionally noted does not necessarily indicate functional disturbance, and in a disease which disappears spontaneously for an indefinite period an apparent cure by electricity or suggestion may well be questioned. Furthermore in few or any of the cases did the symptoms appear to be such as to suggest a functional origin.

Assembling our facts, we may say that ophthalmoplegic migraine is not a morbid entity but rather a peculiar syndrome characterized by irregularly recurring, usually prolonged, migraine-like attacks which terminate in a complete or incomplete paralysis of one or more of the motor nerves of one eye, especially the third nerve. The paralysis, at first intermittent, tends to become later remittent, then permanent. Coincident affections of other nerves, especially the 5th, 7th, and optic nerve, occasionally occur. In a few cases the condition is bilateral, but most cases of bilateral recurring paralysis present a different picture and fall into another category. The cause of genuine ophthalmoplegic migraine

is generally a basal lesion of varying character, but a few cases are probably nuclear in origin.

Treatment of ophthalmoplegic migraine has in general proved futile. Clarke apparently relieved one case by correction of the refraction; and Priestley Smith by dietetic treatment relieved another of the headache but not of the paralysis. Removal of all possible causes of infection (intestinal or otherwise) and improvement of the general condition in every possible way would always be in order. Relief may be afforded in this way. But a case would have to be under observation for a long time before one could with certainty report it as cured.

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FURTHER OBSERVATIONS ON "NEW METHOD OF PREVENTING POST-OPERATIVE INTRAOCULAR INFECTIONS."[1] REPORT OF 1250 SUCCESSFUL CASES.

By Dr. GEORGE HUSTON BELL, New York.

IN presenting to the medical profession my second paper on the prevention of "Post-Operative Intraocular Infections," it is a matter of profound gratification to be able to state that in the 1250 cases herewith presented there has not been a single primary infection or a case of panophthalmitis. This, I think, is a great tribute to the method employed.

In my first paper before the American Medical Association in 1921, my technique was outlined and has not since been changed. The time has come when we can now approach the operating table with a feeling and a confidence, such as we have not had in the past, and I feel that a great load has been lifted from the shoulders of most ophthalmic surgeons.

How often have you witnessed an infection following intraocular operations? Unfortunately, this calamity has overtaken us all. How often have you been told by the bacteriologist that the smear from the conjunctival sac was dirty and how often have you waited for days for the smear to become negative before operating? You then operate and dress the eye with fear and trembling, forty-eight hours afterward. Again, we have been told by the bacteriologist that the smear was negative; we operate and in forty-eight hours, we dress the eye and find an infection—this, I believe, has happened to us all.

[1] Read before the Section on Eye, Ear, Nose and Throat, at the Annual Meeting of the Medical Society of the State of New York, at New York City, May 23, 1923.

In no case of our 1250 did we wait for a single day before operating to clean up whatever was found in the smear from the conjunctival cul-de-sac.

Much valuable time is lost by trying to clean up the conjunctiva when there is a questionable smear. The time that is lost is due to inadequacy of hospital facilities and also to patients. According to my method we do away with all this "lost motion." If a patient's eye looks clinically clean, we go ahead and operate regardless of the findings in the smear. The so-called "senile catarrhs" are not looked upon as clinically unclean by us. But we do exert pressure over the lacrimal sac in such a way as to cause the smallest quantity of fluid present in the sac to flow back through the canaliculi. Cases of mild infection limited to the sac will thus be detected and must be cleared up before we regard the eye as clinically clean. In other words we pay no attention to the findings of the bacteriologist.

I have such strong faith in my method of treatment that I am anxious for all ophthalmic surgeons to give it a trial, unless they have some better plan of procedure.

TECHNIQUE.

First step—The focal infections, such as oral sepsis, diseased tonsils, and toxemias of the intestinal tract, must be removed. I hold that it is just as necessary for the ophthalmic surgeon to get his house in order before subjecting his patient to a major operation upon the eye, as it is for the general surgeon to clean house. Every patient of mine, whether clinical or private, must stand the acid test for the "Three T's" (teeth, tonsils, and toxemia) before we even consider the operation. This routine is carried on in my clinics at the New York Eye and Ear Infirmary, and the New York Polyclinic Hospital, and also in my private practice. I said in Atlantic City at the A. M. A. session in 1919, that "a dirty mouth was one of the greatest menaces to the human race." I wish now to reaffirm and confirm that statement. We must make no mistake about oral sepsis, as it is the arch enemy of the ophthalmic surgeon. The germs themselves and toxins or their poisonous products must be eliminated from the system as far as lies in our power,

if we are to better our cataract results. In this way we can hope to reduce the amount of secondary post-operative infections. If we clean the blood by getting rid of toxemia, then we will raise the resistance of our patients, so that we may be able to approach the operation with a confidence and courage such as we have not had in the past. We must bear in mind that the longer toxemia exists the less nerve energy and resistance there is.

All of this work on focal infections must be done from two to three months before the patient is admitted to the hospital. I lay great stress on this work, as we must study the general state of patients more than we have done in the past. A Wassermann test is made in zonular and complicated cataracts also in hard cataracts, when necessary.

The urine is examined several times previous to the operation, and the height of blood pressure must be known.

Second step—The patient is then admitted to the hospital. Twenty-four hours before the operation, a dose of castor oil is given, as nothing sweeps and cleans the intestinal tract like castor oil. Castor oil has been the standby of physicians since time immemorial. I know of no substitute for it.

Third step—Two hours before the operation, a smear of the conjunctival sac is taken, after which two drops of a 1 per cent. solution (5 grains to an ounce of distilled water) of silver nitrate are instilled into each eye.

The silver produces an irritation, but not an inflammation, so that when the patient comes to the operating table the eyes look red and inflamed, with a certain amount of mucus at the inner corners. The brow, the eyelids, and adjacent skin are washed thoroughly with castile soap and water and the eyes are then washed out with a normal salt solution, as a means of freeing the operating field of mucus, dust, etc. I am now using novocaine (2% solution) paralysis of the orbicularis as a routine, and regard it as a great advance in the technique of extraction. Also it is imperative to have your assistants wear sterile rubber gloves. After the patient is well under the local anæsthesia, the operation is performed. We always try to get a conjunctival flap. Our usual technique is then employed. Afterward we use 2 drops of 3% solution of atropine, and a 25% solution of argyrol, after which both eyes are bandaged for

forty-eight hours. Then every two days, the eye is dressed and argyrol and atropine instilled.

OPERATIONS PERFORMED IN 1250 CASES OF INTRAOCULAR DISEASE.

Case	*Diagnosis*	*Smear*	*Operation*
10	Chronic glaucoma	Pus, mucus, large diplococci	Lagrange
145	Immature and mature senile cataracts	Negative	Prelim. iridectomy
50	Mature senile cataracts	Epithelium, K.L.B.	Prelim. iridectomy
50	Mature senile cataracts	Leucocytes, large and small diplococci	Prelim. iridectomy
75	Mature senile cataracts	Pus, leucocytes and mucus	Prelim. iridectomy
65	Glaucoma	Negative	Iridectomy
25	Acute Glaucoma	Pus, pneumococci, mucus	Iridectomy
25	Old iritis	Mucus, leucocytes, no bacteria	Iridectomy
20	Occluded pupil	Negative	Iridectomy
20	Anterior synechiæ	Negative	Iridectomy
30	Chronic glaucoma	Negative	Lagrange
10	High myopia	Negative	Linear extraction
10	High myopia	Negative	Discission
20	Chronic glaucoma	Pus, cells, mucus	Trephine
15	Capsular cataracts	Occasional diplococci	De Wecker's
15	Secondary cataracts	Pus cells, mucus, bacteria	De Wecker's
10	Postpolar cataracts	Mixed germs, diplococci	Discission
20	Zonular cataracts	Negative	Discission
10	Zonular cataracts	Pus cells, mucus, bacteria	Discission
30	Secondary cataracts	Negative	Discission
30	Secondary cataracts	Mucus, leucocytes	Discission
5	Buphthalmia	Leucocytes, large diplococci	Iridectomy
20	Traumatic cataracts	Negative	Linear extraction
10	Traumatic cataracts	Leucocytes, cocci	Linear extraction
20	Traumatic cataracts	Small, large diplococci	Linear extraction
10	Zonular cataracts	Negative	Linear extraction
20	Congenital cataracts	Negative	Linear extraction
5	Morgagnian cataracts	Mucus, pus, diplococci	Extraction
120	Mature senile cataracts	Negative	Extraction
25	Mature senile cataracts	Pus, cells, no bacteria	Extraction
25	Mature senile cataracts	Leucocytes, occasional coccus	Extraction
10	Mature senile cataracts	Mucus, bacteria, leucocytes	Extraction
30	Mature senile cataracts	Epithelium, pseudo "K.L.B."	Extraction
20	Mature senile cataracts	Much mucus, no bacteria	Extraction
20	Mature senile cataracts	Mucus, diplococci, pus cells	Extraction
40	Mature senile cataracts	Leucocytes, no bacteria	Extraction
40	Mature senile cataracts	Mucus, staphylococci	Extraction
10	Mature senile cataracts	Pus, pneumococci, mucus	Extraction
5	Mature senile cataracts	Mucus, leucocytes, no bacteria	Extraction
10	Mature senile cataracts	Mucus, few cocci, no pus	Extraction
20	Mature senile cataracts	Leucocytes, large and small diplococci	Extraction
20	Mature senile cataracts	Pus, mucus, leucocytes	Extraction
20	Mature senile cataracts	Mucus, large and small diplococci	Extraction
20	Dislocated lens	Mucus, leucocytes, bacteria	Extraction
30	Complicated cataracts	Pus cells, mucus, bacteria	Extraction
4	Cyst of iris	Negative	Iridectomy
6	Detachment of retina	Negative	Trephine

SUMMARY.

If we are to improve our cataract results and have fewer post-operative infections, we must start at the bottom and not at the top. It is just as necessary for the ophthalmic surgeon to clean house, as it is for the general surgeon.

1. The arch enemy of the ophthalmic surgeon is oral sepsis. Patients must stand the acid test for focal infections, the "Three T's" (teeth, tonsils, and toxemia), before we will consider the operation. This applies to all patients except emergency cases. Wells, before the New England Dental Society, called attention, in no uncertain way, to the importance of going after oral and focal infections before attempting operations where the eyeball is opened.

2. I wish to express strong faith in silver nitrate as a germicide and irritant. In all of these 1250 cases, 1 per cent. silver nitrate was instilled into the eye two hours before the operation, and 25 per cent. solution of argyrol was used at the time of operation.

3. If a patient's eye looks clinically clean we operate. The so-called "senile catarrhs" are not looked upon as clinically unclean by us. We pay no attention to the findings of the bacteriologist. In fact, in private patients, I no longer take smears.

4. In analyzing our 1250 cases, we have three secondary infections, coming on nine days after the operation. One of these patients recovered and will have some vision: the other two developed chronic iridocyclitis. These patients had negative smears. We were able to trace these infections to faulty technique in reference to oral sepsis.

5. Case 1 and 2 occurred in the same patient. His smear showed pus and diplococci, mucus; also he had chronic dacryocystitis. The patient had chronic glaucoma with very little vision. I performed a Lagrange operation without bringing the tension down; in two weeks, another Lagrange operation was performed. No infection followed either of these operations. Doctor Stout was house surgeon at that time and assisted.

6. Vitreous humor was lost in four cataract cases. In case 55 a bead of vitreous humor appeared. The smear contained

pus and a number of diplococci. No infection resulted. Case 167 was a bad case of ozena; a smear showed pus cells and staphylococci. There was no infection. An iridectomy was performed in Case 280, after which a secondary membrane was removed. Some vitreous was lost. A smear showed considerable mucus and a few leucocytes and an occasional diplococcus. There was no infection. In Case 391, an iridectomy and capsulectomy were performed. Vitreous was lost; the smear showed pus, diplococci and mucus and no infection resulted. Case 518 presented a dislocated lens. Iridectomy and extraction of the lens were performed; considerable vitreous was lost in this case; the smear showed a few leucocytes and some bacteria—no infection resulted. In case 799, there was ozena. A smear showed leucocytes and large diplococci. No infection resulted. In case 897, the patient squeezed and lost considerable vitreous. The smear showed epithelium and pseudo K.L.B. No infection resulted.

7. Case 1150—Mr. H., age 68, with mature senile cataract, was referred to me by Doctor Emil Mayer. This patient had been troubled for years with an obstruction of the nasal duct. Probes had been passed into the duct from time to time. The canaliculi were still open, but no pus could be squeezed out of the sac. He had angina pectoris and would not submit to extirpation of sac. Doctor Mayer and I decided to undertake the extraction of the cataract with an extra dose of silver. One per cent. silver nitrate solution was used in his eye at 9 A.M. and again at 12 o'clock. At 2 P.M. the operation was performed, the patient sitting up in a chair on account of his heart. His eye was dressed every 24 hours on account of his lacrimal trouble. He got a beautiful eye with $\frac{2}{18}$ vision. The smear in his case, before the silver was instilled, showed pus cells, diplococci and mucus—but for its "watery" condition the eye looked clinically clean. Doctor David Robb was house surgeon on my division at "The Infirmary," and assisted me in this operation.

Case 1200 was a similar case in a woman, age 60, with lacrimal trouble of long duration. Her smear was negative and her eye looked clinically clean. I operated upon her as I did on Mr. H., using silver nitrate 1% at 9 A.M., and again

at 12 o'clock, and extracting the cataract at 2 P.M. Her eye was also dressed every day. Doctor H. V. Halbert was House Surgeon at the time, and assisted me. I mention Case 1, 2, 1150 and 1200 especially to show to you and to prove to you the great power silver nitrate has as a practical specific against infection. Of course, I am not advocating operating upon every cataract case that comes along which has serious trouble with the lacrimal apparatus without first performing an excision of the sac. But I do think, if the eye looks clinically clean and no fluid or secretion can be squeezed out of the sac, and 1% silver nitrate is used twice in the eye, and at four-hour intervals, it is perfectly safe to operate. Investigations made by Elsching and by other workers have proved that in the majority of cases, the conjunctival sac contains the pathogenic organisms.

8. In soft or lamella cataracts, I perform a peripheral linear incision with a flap just back of the limbus. Also in all discissions, I enter the anterior chamber through the conjunctiva and sclera, just back of the limbus. The same rule applies to hard cataracts. I make the section in the sclera just behind the limbus, always having in mind a conjunctival flap. I consider it *obsolete* to go through the cornea in performing discissions, soft or hard cataracts. Why attack the eye at its weakest point? The chances for infection are greatly increased by using the corneal route.

9. In our list of 1250 cases we operated upon 415 mature senile cataracts, 80 soft cataracts, and 30 complicated cataracts —making all told, 525 cataracts removed, with two eyes lost from secondary infections coming on nine days after the operation. Not a single case of primary infection or panophthalmitis resulted. We performed 125 discissions on soft and secondary cataracts, 480 iridectomies, 40 Lagrange, and 26 trephine operations, 30 DeWecker, and 24 miscellaneous operations without a single primary or secondary infection.

It is interesting to note that the ages of our patients ranged from two months to ninety years. Eight of our patients with cataract had three per cent. sugar in the urine. Sugar is no barrier to the operation unless it exceeds four per cent.

Duane (in seventh edition of Fuchs, page 846) in speaking of my method of preventing infections calls it "Artificial Pre-Operative Leucocytosis," and he further adds: "The good results obtained by this practice in eye operations seems well substantiated by statistics and the translator believes that it is based on sound theory."

ACTION OF SILVER NITRATE.

I know that silver nitrate furnished a marked degree of stimulation to the conjunctiva and in that way leucocytosis is produced. The silver promotes phagocytosis and the formation of antibodies, and in this way puts the tissues into a stronger state of defense against the invasion of micro-organisms. Even were an inflammation produced by the silver, it would only diminish the probability of infection, not increase it, because the leucocytes and their leukins are valuable means in combating bacteria. And what makes the phagocytes so active? It is the pre-operative removal of foci of infection—going after the "Three T's."

The thought has occurred, however, that this preventive effect of the silver salt may be at least in part due to the germicidal action of the silver, and in order to shed more light on the subject a series of 50 cases have been investigated by Doctor S. P. Oast and Mr. E. B. Burchell in the laboratory of the New York Eye and Ear Infirmary.

The following technique was employed on the patients admitted to "The Infirmary." A culture was taken from the conjunctiva. Then according to the author's technique, several drops of freshly prepared silver nitrate in 1% solution were instilled into the lower conjunctival cul-de-sac, and the eye left unprotected by a dressing of any sort. After a lapse of two hours, just before the operation, a culture was again taken in the same manner. The material was collected by means of a small platinum loop from the lower conjunctival cul-de-sac, care being taken to avoid contamination with the lids and cilia. This was transferred after the usual manner to agar starch which was incubated at 37° C. for twenty-four hours and then examined.

BACTERIOLOGICAL STUDIES.

No. cases examined	*Before*	*After*
3	Diplococci	No growth
3	" and Xerosis	No growth
4	Xerosis	No growth
5	Staphylococcus	No growth
1	Subtilis bacillus	Staphylococci
1	Xerosis and Diplococci	Xerosis and Diplococci
4	Staphylococcus	Staphylococcus
1	Xerosis and Staphylococcus	No growth
1	Xerosis	Xerosis
1	Streptococcus and Staphylococcus	Staphylococcus
1	Xerosis and Staphylococcus	Xerosis
23	No growth	No growth

We concede the generally accepted fact, that from a practical standpoint, it is impossible to sterilize the conjunctival cul-de-sac, and were this possible of accomplishment, even momentarily, by any anti-bactericidal agent, or method, at our command, as soon as the same had become neutralized or its action spent, infection would immediately recur from the lid margins and meibomain ducts which are constantly contaminating the conjunctiva with their germ-laden secretions. It will be noticed therefore, from the above study that there is but one case which shows, after the use of silver, the presence of any organism (diplococcus), not a normal inhabitant of the conjunctiva, or whose presence could not be accounted for by contamination in the above mentioned way. Close analysis of the above results will also show that in some of the cases different organisms were found before and after the use of silver, a circumstance which likewise adds weight to the view of constant contamination in the above-mentioned way.

The older ophthalmic surgeons and writers were extremely averse to the use of any actively germicidal agents in the conjunctival cul-de-sac just prior to the intraocular operations because of the usually attendant irritating qualities of such materials. They feared the catarrh produced by this irritation with its supposed concomitant increase in the number of bacteria, thereby favoring rather than diminishing the chances of infection. In the light of these present studies, not to mention the corroboration of our clinical experience, there

seems little ground for such assumption as regards at least one agent which is capable of very appreciable germ destroying power in vivo.

In the present series in cultures taken two hours after the use of silver, in every case, a point was made of obtaining some of the stringy purulent-like masses such as are familiar to all who have observed the effect of silver solution in the eye, and it was extremely interesting to observe that the cultures obtained in this way invariably yielded no growth, or a considerably diminished number of colonies. In addition to the silver being a powerful bactericide, I am strongly of the opinion that it increases the phagocytic power of body cells in and around the eye, producing a leucocytosis and leaving the eye protected during the healing process.

PREPARATION OF "THE SILVER."

The 1% solution of silver nitrate (5 grains to 1 oz. distilled water) should be made up fresh at least every three months, and must be kept in a dark glass-stoppered bottle. The rays of light cause the silver solution to become inert, and for that reason it should also be kept in a dark room.

Professor P. Knapp in an article (*Schweizerische, Medizinische Wochenschrift*, April, 1923, Basel) on Modern Cataract Operation, makes several observations concerning the use of $AgNO_3$.

First he refers to the instillation of 1% to 2% silver nitrate immediately before the operation. The point of using the "silver" two hours before the operation, which is most important, he has overlooked. The two-hour interval gives the silver time to destroy bacteria and produce a leucocytosis.

His second objection was that the silver increases secretion with many bacteria. Our research work at the New York Eye and Ear Infirmary shows that the silver does increase the secretion but cultures of the stringy purulent-like masses showed that they invariably yielded no growth or a considerably diminished number of colonies. In other words, two hours after the instillation of the 1% silver nitrate in the eye, cultures from the secretion of cul-de-sac showed the absence of the bacteria in 82% of the cases, whereas before the silver

was instilled, the cultures were positive in 60% of the cases.

I feel that Professor Knapp is making the wrong application of a good principle. If he will use my technique with an open mind, I feel that he will be pleased with the results. Certainly my clinical results are overwhelming.

The following well-known ophthalmic surgeons in Greater New York are using "My Method" in their intraocular operations. W. E. Lambert, J. H. Ohly, P. C. Jameson, C. E. McDonnald, J. M. Wheeler, F. W. Shine, L. W. Crigler, A. Wiener, I. Hartshorn, B. W. Key, C. Berens, Jr., B. Samuels, E. V. Darling, T. A. Northcott, and J. H. Dunnington, I. Goldstein and K. Schlivek.

I want to express my thanks to the following members and ex-members of the House Staff for their coöperation and assistance:—Anthony, Peters, Stout, Blades, Richardson, Neff, Hicks, Shannon, Robb, Hogan, Hinsdale, Pierkey, Veasey, McGarvey, Oast, Frost, Lewis and Halpert. Also I want to express my thanks and great appreciation for Doctor S. P. Oast and Mr. E. B. Burchell for this excellent research work.

In addition to the 1250 cases—the subject of this paper, R. C. Heflebower of Cincinnati reports 300 intraocular operations performed according to "my method" without a single primary or secondary infection. Heflebower feels such a sense of security with "The Silver Nitrate," that he now no longer takes smears or cultures—reliance being placed in the efficacy of the preparatory treatment, and he adds, "The results have been most happy."

PRELIMINARY IRIDECTOMY OF PRIME IMPORTANCE.

1st—It is much safer for the patient's eye and better for the surgeon in the long run.

2nd—It protects the eye and leaves it stronger for the years to come.

3rd—If it is the "safe procedure" when a patient has one eye,—why is it not a good thing when he has two?

4th—Doing preliminary iridectomies in my clinic and in my private practice have become a "hard and fast rule" with me. This rule applies to all (hard, soft and juvenile) cataracts. It enables me with safety to the eye to do all my operations just

back of the limbus. I feel that it is a great mistake to use the corneal route in doing intraocular operations.

5th—Some operators and a few patients raise the objection to iridectomy on account of the necessity of making two trips to the hospital and because the danger of infection is doubled by a two-step operation. W. F. Hardy says, and he is quite correct, that the operator raises these objections in his own mind much oftener than does the patient, and he adds "if an ophthalmologist is thoroughly convinced that a certain line of action is the correct one, he will have little trouble in putting that conviction over to the patient and gaining his consent." As far as the extra chance for infection is concerned, I feel that I have been able to eliminate that argument.

6th—I agree with Darrier and Hardy who are strong advocates of preliminary iridectomy.

7th—If you want every cataract operation to be successful, then I advise "my technique," and a preliminary iridectomy. I hold and believe that it is the only way to get a good surgical result in every case. These are the conclusions that I have reached after doing cataract work for over twenty years.

THE TREATMENT OF SOME FORMS OF DERMOID AND OTHER CYSTS WITH TRICHLORACETIC ACID.

By Dr. H. GIFFORD, University of Nebraska, Omaha.

SEVERAL years ago, in discussing the treatment of blood cysts of the orbit,[1] I stated that if I should again encounter one of those dermoid cysts which send a prolongation of the cavity deeply into the orbit, I should be inclined, instead of trying to dissect this out, to destroy its epithelial lining with trichloracetic acid. In the intervening period I have had five cases of dermoid cysts, and two so-called oil-cysts (like the one recently reported by Arnold Knapp),[2] in which this treatment has been tried with perfect success.

Case 1. Robert P., aged 19; has noticed a tumor in the upper angle of the right orbit for many years but he thinks it is slowly getting larger. It is not painful but he notices that it seems larger when he catches cold. Examination shows a tumor nearly an inch in diameter just above the tendo-oculi. This recedes slightly on pressure but on deeper pressure is found to be firmly attached to the inner wall of the orbit and to be free from the superior wall. Examination of the nasal cavity shows nothing noteworthy. Under general anæsthesia, the skin was incised and a fixation thread was passed through the wall of the tumor. The latter was then opened, revealing a cavity about one inch in breadth and one and one-half inches in depth, full of broken down epithelium. This was swabbed out carefully and liquified full-strength trichloracetic applied freely and thoroughly to the interior of the sac. The external wound was completely closed and healed without any reaction. Seven months later he writes that the swelling is practically

[1] *American Journal of Ophthalmology* (New series), Vol. i., p. 625.
[2] *Archives of Ophthalmology*, Vol. lii., No. 2, March, 1923, p. 163.

gone and the scar is hardly noticeable. He also adds that a lacrimation which had bothered him for years (but which he had not mentioned on his first visit) has entirely disappeared. He was urged to write me again if there were any signs of a recurrence at any time, but I have not heard from him since.

Just as good a result *might* have been obtained by excision but this would have been decidedly more difficult and would have involved some risk of injuring the superior oblique.

CASE 2. A woman about twenty years old (whose case card I have not found on account of faulty indexing) came to me for a tumor about three-fourths inch broad, extending from the zygoma for about two inches straight down, immediately in front of the right tragus. This was rather closely attached to the skin but deeper in it was tolerably moveable. I felt that there would be some risk, in dissecting it out, of injuring some vessel, nerve, or duct, without advantage to the patient; so I made a horizontal incision about one-fourth inch long at the upper extremity of the tumor and evacuated a large quantity of characteristic dermoid substance. The cavity was then treated as in Case 1, several drops of the liquified trichloracetic being injected deeply down into the cavity with a long-pointed dropper, before the swabbing process was begun. The wound healed without reaction and I have heard nothing more from her during the ensuing years. This I take to be a good sign as she lives only a few miles away.

Three other rather superficial dermoids from one-fourth to one inch in diameter have been treated in the same way, with perfect results in two, while in the third, the wound failed to close by first intention, and a slight watery discharge persisted for several days, the eventual result being perfectly good. The last three tumors might just as well have been excised, the last one, in fact, being good evidence that where the cavity does not penetrate deeply, the excision is preferable.

CASE 6.—Martha C., aged 19; lost left eye when three years of age from a cut with glass. For the past two months has noticed a growth in the upper inner angle of the left orbit which has pushed the artificial eye out and caused headaches. On examination a tumor apparently about one-fourth inch in diameter was found in the upper inner angle of the orbit. An incision was made through the skin about five-eights inch long, exposing a cavity about one inch in diameter, filled with a slightly syrupy, perfectly clear, light straw-colored fluid. This led back along the inner wall of

the orbit but no bare bone was found nor any connection with any other cavity. Trichloracetic acid was applied and the cavity packed with aristol. On the fifth day the wound was sutured, although there was still a good deal of aristol in the cavity; and she returned to her home in a neighboring city. Her family physician wrote me, two weeks later, that the stitches came out after a few days and a good deal of pus came from the cavity for several days but then it healed completely, with a very inconspicuous scar.

The operation was done a year and a half ago and she has had no further trouble. I must confess that I cannot remember why I did not close this wound without packing, as I had done in the other cases.

CASE 7.—This case is practically a twin to Case 6. The patient Miss M. L., aged 28, has a tumor at the inner angle of the right orbit. This was noticed when she was a year old and has been very slowly increasing in size. Has been somewhat painful at times. Three years ago she was struck in this region with an elbow, after which it has increased more rapidly.

Examination shows a small xanthelasma in the skin of the upper inner orbital region on the surface of a roundish tumor about three-fourths inch in diameter. This is firm and elastic and does not reduce on pressure. Tear passages normal. The tumor transilluminates readily when light is thrown through the skin at the side. Tumor was opened and proved to be a so-called oil-cyst, precisely like that in Case 6. There were no signs of the ordinary epidermal débris except at the extreme inner end of the cavity where epithelial-like stuff showing no structures under the microscope, was dug out with a vigorous application of a swab. This was somewhat pinkish, not of the yellowish-white color found in dermoids. The cavity was about an inch in depth and three-fourths inch in diameter, reaching along the inner wall of the orbit to which its wall was closely attached. This was swabbed as clean as possible and the cavity packed to stop bleeding. Packing then was removed and several drops of trichloracetic acid injected into it; the walls thoroughly scrubbed again with the acid; a little aristol was introduced into the cavity and the opening closed with three sutures. Healing was by first intention. When seen May 1, 1923, the wound was hardly perceptible and she had had no pain since the operation.

The idea of closing dermoid cysts by destroying their lining is not new. So far as I can learn the only substances that

have been used for this purpose have been tincture of iodine, nitrate of silver, or a mixture of iodine with 10% carbolic. The rare references which I have found to this treatment in text books are apparently based more upon tradition than upon experience. The great majority of books which I have consulted speak merely of excision, under the head of treatment.

Conclusions.—Dermoids and other cysts, the excision of which involves some risk of injuring important structures, may safely and effectively be treated by destroying the lining with trichloracetic or some other caustic. Where such cysts are entirely superficial, simple excision with thread fixation is to be preferred. There is no reason why the cauterization method should not be combined with excision, as I suggested in my blood-cyst paper; the prominent, easily accessible portions of the cyst being excised, while the remainer is cauterized.

CONGENITAL OCULO-FACIAL PARALYSIS.[1]

By Dr. DANIEL B. KIRBY, New York City.

THE complexities of the oculo-motor mechanism would easily admit of a great number of congenital defects. However, cases showing agenesis or abiotrophy of portions of this apparatus are comparatively rare. It is our purpose to present a patient with bilateral oculo-facial paralysis, most probably congenital, and to review a few analogous cases.

Patient, male, age 52, has never been able to close his eyelids nor to move his eyes to either side. He is the youngest of ten children, all others being entirely normal. Birth was normal but the inability to close the eyes, or to move the eyes to either side and the expressionless face were noted from early infancy.

Twelve years ago he contracted lues and did not receive proper treatment. In November, 1922, because of ataxic condition, he fell down a flight of steps and suffered lacerations about the left eye and fracture of the outer plate of left frontal sinus. This brought him to my care at Bellevue Hospital on the service of Dr. Charles H. May. The lacerations healed rapidly and no untoward result came from the fracture. The presence of lues was confirmed by Blood Wassermann 2+, Spinal Fluid Wassermann 4+, Colloidal Gold 0001221000. The interesting muscular anomalies were noted and diagnosis confirmed while in the hospital by Dr. Foster Kennedy.

He has been receiving anti-luetic treatment since and the tabetic symptoms have abated somewhat, but otherwise he presents the same phenomena now that he did then.

Status Præsens—

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[1] Case presented before Ophthalmic Section, New York Academy of Medicine, April, 1923.

He is unable to elevate his brows. He is unable to close his eyelids. On the attempt to do this, the globes are rotated upwards and inwards. There is marked ectropion of both lower lids. With the eyes directed at a distant object there is present, esotropia of about 10° of arc with right hypertropia of about 3° of arc with the left eye fixing. When the fixing eye is covered, the right eye will take up fixation, intorsion and depression being observed. When the left eye is released from cover, it will resume fixation by elevation and extorsion.

There is no evidence of action of either external rectus. Patient is unable to rotate either eye outward. Vestibular tests carried out by Dr. J. Swift Hanley indicated activity of both labyrinths but did not produce any external rotation of globes nor any nystagmus in the horizontal plane.

When commanded to move his eyes from side to side, the patient is unable to move them. When an object is carried in a horizontal plane at a distance of 20 feet, he is unable to follow it with either eye, when the head is fixed, only shift of fixation from one eye to another at the mid-line being noted. When the object is carried at near range to the right, the left eye follows it through an arc of 10°. To the left, the right eye does likewise. When a distant object is fixed by the eye and the head passively rotated to the right, the right eye maintains fixation through an arc of 25°. On rotation to the left the left eye does likewise through an arc of 20°. Similar results for near range. There apparently is, in this case a greater ability to move the eyes in response to lower reflex control of associated movements than to those of the higher centers.

The patient is able to converge although this power is less than normal. He is unable to relax this convergence and to return to his primary position. Inasmuch as all associated movements within near range have also the added phenomenon of convergence, it was necessary to differentiate. While one eye was occluded by a screen and the other eye was called upon to follow the object in movement of adduction, the covered eye remained stationary. Both pupils reacted to convergence in the act. The same was true when the opposite eye was tested in like manner. He can elevate each eye and depress them normally in a single vertical plane concomitantly and without any torsion. Diplopia has never been experienced, nor has it been induced.

Pupils are unequal, irregular, do not react to light, either direct or indirect, but do react to accommodation, both monocular and binocular, and to convergence.

Media show a few vitreous opacities. Fundi-disks ill-defined, slightly paler than normal, no abnormal elevation

nor depression. Slight evidence of vascular thickening. Fields show slight concentric contraction.

Other neurological findings as found by Dr. Foster Kennedy at Bellevue Hospital and by Drs. Wechsler and Rubinowitz at Vanderbilt Clinic were: I, VIII, IX, X, and XI cranial nerves normal. VII showed a bilateral facial atrophy of the greater number of muscles supplied by both the upper and lower divisions of the facial nerves. Electrical tests conducted by Dr. Evan Evans elicited no response to any form of stimulation in the right or left frontales, corrugatores, orbiculares oculi or orbiculares oris, quadrati labii inferioris, or of the menti. The right and left quadrati labii superioris and right and left zygomatici responded to 0.4 M.F. The XII showed a bilateral paresis. The tongue deviated to the left, cannot be moved upwards. Lateral movements fair. A deep median furrow with other minor furrows indicated atrophy of some of the intrinsic muscles of the tongue, especially the superior longitudinal fibers. Direct stimulation of the tongue gave response to 0.4 M.F. Talipes Equino-varus of moderate degree on the right side.

Evidence of neuro-lues was shown by the following: Argyll Robertson pupils, absent patellar and Achilles tendon reflexes, ataxia of lower extremities with positive Romberg, variable tactile sensory changes in lower extremities.

Summary.—History of inability to move eyes to either side, or to close the eyes and difficulty with movements of tongue from earliest period of life with later contraction of lues in 1911 and onset of tabes dorsalis.

Etiology.—The neuro-lues needs no discussion here, but the earlier condition most probably represents an agenesis or abiotrophy of the several cranial nerve nuclei. It is necessary also to consider an inflammatory process occurring very early in life either in the nature of an encephalitis or vascular injury in the region of the VI nerve nuclei, affecting the contiguous loop of the VII nerves, with an affection of the XII nuclei and possibly the posterior longitudinal bundle, but the peculiar distribution militates against such a cause.

In 1897 Leszynsky (1) presented a case of congenital absence of outward movements of both eyes but with retention of

convergence and of lateral adduction of either eye. He referred to an analysis by Kunn (2) in 1895 of nine cases of bilateral abducens paralysis, and with few exceptions these were associated with bilateral facial paralysis of the nuclear or infranuclear type.

Cases which were analogous to that of the author, *i.e.*, in presenting paralysis of both abducens and facial nerves with other features have been reported by:

Graefe (3), whose patient, a male age 20 years, showed also absence of sense of smelling and taste.

Kahlmeter (4) has recorded a case of bilateral facial and abducens paralysis as also did Harlan (5) in the case of a boy 18 years of age without remarking on any additional features.

Chisolm (6) observed a woman age 35 who showed in addition, the absence of both carunculæ lachrymales.

Möbius (7) has written of a man 59 years old who also showed paresis of both inferior recti and the presence of webbed fingers.

Fryer (8) has given us the case of a youth of 16 who had also absence of associated lateral movements, absence of the right hand and incomplete development of the right upper extremity.

Schmidt (9) recorded the case of a boy 6 years of age who had also disturbances in the hypoglossal nerve; the fact that the recti interni acted well in convergence and in lateral adduction was also remarked.

Procopovici's (10) case of a girl age 7½ years showed the absence of the facials and abducens but was able to use the interni well in convergence and in associated lateral movements.

Lennon (11) has given us two cases, one of a male child 2 years old who had also diastasis of the abdominal recti muscles, and the other, a boy age 14 with also club feet and hypoplasia of the right arm and hand.

Economo (12) reported a case of Güterman's, a boy of 6 with also associated paresis of the lingual portion of the right hypoglossal, with epicanthus and malformation of caruncles, trunk and fingers.

Warrington (13) presented a case which showed absence of function of both abducens but with a unilateral facial paralysis.

Not entirely analogous but sufficiently of interest to be

recorded in this connection are cases of congenital absence of both external recti, without loss of function of the facials as in the cases recorded by Leszynsky (1), Lamhofer and Möbius (14), Günsberg (15), Kunn (16), and MacKinlay (17), the last mentioned recording also that the function of convergence was retained.

In this connection also should be recalled those cases exhibiting the bilateral retraction syndrome due to the congenital absence of muscle tissue in the external recti and its replacement by fibrous tissue. This subject has been ably reviewed by Duane (18) in 1905 and more cases are continually being recognized and found in the literature since attention was directed to them.

Etiology.—In regard to the underlying causes of such cases as are analogous to the author's, different opinions have been expressed, not however contradicting each other, but indicative of the conditions found in the individual cases.

The two main classes may be considered as

(a) Those in which there exists a primary aplasia of the muscle, due to a mesodermic defect, the embryonic foundation for the muscle not being laid down. There is sufficient evidence that this occurs, for Schenkl (19) as quoted by Lennon, (11), reported a case of ophthalmoplegia, in which the muscles were absent, while the nerves and ganglion cells were intact at autopsy. Obersteiner (20) examined the cord of a man who lacked from birth, muscles of the right shoulder girdle and at post-mortem examination found intact corresponding anterior horn cells. McDannald (21) and Mittendorf (22) have recorded cases of absence of ocular muscles as demonstrated at time of operation.

(b) The other cause considered is an ectodermic defect. 1. Those cases in which the muscle was laid down but underwent degeneration during the embryonic period because the normal connection with the nerve or central organ was never established. 2. Lack of development of the nerve. 3. Lack of, or defective development of the cells of the nucleus.

Gowers (23) referred to this class of cases and without pathological evidence at the time, stated that the history suggested defective vital endurance in the nuclear structures.

He placed them in the class of abiotrophy as later defined by Collins (24).

A case which was inferred to be central was cited by Lagrange (25) who by carefully tested electrical reactions, demonstrated that the seventh nerve lesion was central and deduced from this fact that the abducens-paralysis was likewise central. Electrical reactions have not as yet helped to solve the location of the lesion in ocular muscle paralysis.

A case in which the cause has been clearly demonstrated to have been due to aplasia of the nuclear structures has been cited by Heubner (26) who in 1900 described a child age 2 years, with paralysis of both external recti and facials and atrophy of the left anterior half of the tongue. Histological examination showed complete absence of the cells of the abducens nuclei. The facial and hypoglossal nuclei on the left side were practically absent and on the right side the facial nucleus was represented by a diminished number of cells. The nerve roots were very small or absent. The left posterior longitudinal bundle was poorly developed. The conclusion was that the lesion was due to nuclear aplasia or agenesis.

In regarding the author's case as congenital, care has been exercised to consider also paralyses occurring after birth either due to abiotrophy as defined by Collins (24), the condition in which a structure apparently normal at birth degenerated through lack of vital endurance, as also the cases of paralysis occurring early in life, as for example, the case reported by Thompson (27), female age 2 years, whose right eye suddenly turned inwards at the age of 18 months, and was followed by the turning in of the left eye ten days later. Paralysis of the right facial was observed in ten days and two weeks later inability to open the mouth. At the time of the report there was present, complete paralysis of both abducens and of both facials, affecting more greatly the lower facial group. Wilbrand and Saenger (28) have demonstrated another case in which there was degeneration of the right abducens nucleus and of a group of cells lying between this and the right seventh nucleus.

However, the absence of the anterior horn cells or of the cells of the cranial nerve nuclei is not absolutely conclusive of nuclear agenesis, as consecutive atrophy of the nerve and

anterior horn cells may occur as was found by Edinger (29), (quoted by Lennon) in the case of a man who suffered an intrauterine amputation of the forearm. Möbius held that neural atrophy in this type of ophthalmoplegia was secondary to the muscular defect, but subsequently changed his view.

SUMMARY.

1. Congenital oculo-facial defects occur
(A) due to mesodermic defect or aplasia of nucleus or nerve or (B) lack of union of nerve with muscle.

2. The nuclear defect has been demonstrated as existing in Heubner's case and others.

3. The author's case probably is analogous and represents an agenesis of the nuclear structures of the VI, VII and XII cranial nerves.

Acknowledgments are gratefully made to Doctors Alexander Duane and John M. Wheeler and to others whose names appear in the paper for their assistance in studying this case.

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MY EXPERIENCES IN WORKING WITH DR. BARRAQUER IN BARCELONA.[1]

By Dr. WILLIAM McLEAN, New York.

THE first writings by Dr. Barraquer in relation to his operation which he has named Phacœrisis, that I have been able to discover were in 1917. Since then he has written quite extensively both in Spanish and in French. When I first read his articles I was much impressed with his manner of extracting the crystalline lens, as previous to my work on the tonometer, I spent considerable thought in an effort to devise a method of removing the hard cataractous lens, employing traction instead of the prevailing method of expression. My thoughts lead to the employment of suction, but I was not considering removal of the lens in its capsule, and I had not successfully worked out a plan along that line before I became interested in an effort to perfect the prevailing methods of tonometry.

Dr. Barraquer was not the first to endeavor to make obsolete the removal of the cataractous lens by expression. Since the early days of cataract extraction we find, creeping into ophthalmic literature, descriptions of methods of operating which would avoid the process of expression in removing the lens from the eye. Pagenstecker attempted to use a curette to lift the lens from the eye, and others have tried to get away from the element of expression in cataract work.

When Dr. Barraquer was in this country last April I had the good fortune of meeting him through the courtesy of Col. Elliot, and Dr. Barraquer consented to demonstrate his operation before the staff of the New York Ophthalmic Hospital. It was my privilege to assist him at one of the operations.

[1] Read before the Brooklyn Ophthalmological Society, Dec. 21, 1922.

This gave me the opportunity of seeing the operation at close range and also demonstrated to me the folly of attempting to master the technic of phacoerisis without the guidance of the author to carry me safely over the many little intricacies and pitfalls, the avoidance of which tends to make for the success of the operation. Only when one works with Dr. Barraquer in Barcelona can he realize the dangers lurking in his pathway had he attempted to work out successfully the technic of Dr. Barraquer. Dr. Barraquer was kind enough to urge me to make the journey to Barcelona where I could avail myself of his teaching facilities, so last summer I spent three weeks working with him.

Dr. Barraquer is Professor of Ophthalmology in the Medical Department of the University of Barcelona, and as such he is Chief of the Dept. of Ophthalmology of a very large municipal hospital. He has access to the autopsy material for ophthalmic purposes of this very large general hospital and it is possible to obtain the eyes of all the patients who succumb in the hospital.

The method of teaching he employed with me was to utilize the freshly enucleated eyes of the recently deceased patients. Because of this wealth of material he has ample good teaching facilities and in this manner I was able to follow his instructions and, by his criticism, develop a technic satisfactory to him. It was not until then that I did operations on the living. Instruction was given in every little detail, and it is the details in this operation that spell either success or failure.

The cataract patients coming under Dr. Barraquer's service are examined very carefully before operation. Urinalyses and physicals are done on each patient. The eye and its adnexa are examined and the type of cataract determined. By type, I mean, not the hard or soft, nuclear or cortical designations as we are accustomed to classify them, but the hard, smooth, evenly grained types, those containing soft areas, and those containing the so-called vacuoles. This designation is possible only with the corneal microscope and slit lamp, and it is with this apparatus that Dr. Barraquer determines the degree of vacuum or negative pressure to employ. The vacuum ranges from 45*cm* of mercury in the softest types to 65*cm* in the hardest cataracts. The hardest and most evenly grained types

of cataract are the easiest to extract, and are those in which the best results are obtained, other conditions being equal.

I have used the term "corneal microscope" to designate this binocular microscope used in connection with the slit lamp. It is true that that is the term commonly used for this microscope and when it was designed, the examination of the cornea was its chief use. After Professor Gulstrand designed and perfected the slit lamp the uses to which this binocular microscope could be employed increased greatly. The iris and lens structures may be studied excellently, and with the assistance of "contact glasses" the angle of the anterior chamber and the vitreous body and retina may be studied under considerable magnification. Because of these added uses to which this microscope may be put, I believe we should drop the word "corneal" and call this instrument a "binocular microscope" since it is more than a "corneal microscope." Its binocular feature has considerable advantage over a monocular microscope, as it gives a sense of perspective when examining the interior of the eye.

With sufficient magnification the crystalline lens assumes an entirely different appearance than when examined with the ophthalmoscope or loop. The mature or immature cataracts as judged by oblique illumination assume very different aspects. The lens which we judge mature and ready for extraction by the Graefe method, when seen with the microscope and slit lamp may present a smooth evenly grained appearance, and as such the highest degree of vacuum applicable would work best. If this mature cataract should present small vacuoles, the degree of vacuum should be regulated to a lessened amount. When large liquifaction spaces are seen in the anterior segments of the lens close to the capsule, a minimum of vacuum that will hold the lens in the grip of the ventouse is used. This minimum ranges around 45*cm* of mercury.

The apparatus by which the vacuum is maintained is a rotary suction pump which is run by an electric motor. The pump runs in a bath of oil, and to the outlet of the pump is attached a piece of rubber tubing the walls of which are of sufficient resistance to prevent collapse. The terminal end of this tubing is attached to the handpiece. Along the line connected to this tubing is a vacuum gauge and a regulator

to govern the amount of vacuum. The hand piece or ventouse is an important part of the apparatus. It contains a valve which is under the control of the ball of the thumb. The tip of the ventouse is of platinum tubing, and the end of the tip is shaped like a tiny spoon with a reinforcement along its edge. The surface of this reinforcement is of the same curvature as the anterior surface of the crystalline lens.

It is essential that the whole suction apparatus be working in perfect order. The valve of the ventouse must not leak, and the guage must be registering a constant steady vacuum of the proper degree for that individual case under operation. In an apparatus where the valve is leaking, suction will occur before the tip of the ventouse is in proper position, and also in such a case, where the regulator is adjusted to maintain correctly the proper vacuum, the vacuum will be increased above the proper amount as soon as the ventouse is applied and the valve opened. Such an improperly acting valve might result in a ruptured capsule as soon as the suction is applied.

Another important feature of a correctly operating pump is to have the strength and rate of vibrations just sufficient to rupture the zonula at the periphery of the crystalline lens. If the vibrations imparted to the air column at the tip of the ventouse are too slow or too strong, traction will be transmitted along the length of the zonular fibers to the ciliary body. Such traction occurs during the Smith-Indian and Knapp intracapsular operations, and Dr. Barraquer believes this traction to be a deterrent factor in these operations. Dr. Barraquer has arranged the stroke of the piston of the pump and the speed just sufficient to break the crystalline end of the zonular fibers. We examined several extracted lenses to make sure that the rupture of the zonule was occurring at the proper place. When others alter the technic of Barraquer by using a tip without a valve or by moving the valve a long distance from the tip so as to use a foot operated valve, an alteration occurs, and operators should study the results of such a change in technic to be sure that the correct principle is still operating in the altered apparatus.

I noticed the description written by another Spanish oculist, Dr. F. Munoz Urra, in a French journal, of an ingenious apparatus by which he imitates the vacuum of the Barraquer

pump. The principle involved in this apparatus employs the expansive and contractive forces of heat and cold. This comparatively simple apparatus consists of a metal tank which is partly filled with water. To the outlet of this tank is fitted a rubber tube with stop-cock. With this stop-cock open the tank is heated by a spirit lamp until the water boils vigorously. The stop-cock is now closed and the tank plunged into cold water. The contractile force of the cold produces a vacuum sufficient to perform the operation of phaccerisis. This apparatus does not utilize a prominent feature of the pump, namely, vibration.

Dr. Marbaix of France, to avoid the slight noise of the pump, produces a vacuum by previously exhausting the air from a comparatively large chamber. A manometer which registers the state of vacuum is connected to this chamber. In this apparatus also the vibration is absent.

Dr. Gallemaerts of Brussels made some experiments in answer to the question raised as to whether the vibrations produced by the pump were carried to the crystalline lens. These experiments carried out in a physics laboratory, by tracings on a moving smoke drum, proved unquestionably that a vibration of the crystalline lens does occur. Other experiments have proved that this vibration breaks the zonule at the crystalline end of the fibers.

Dr. Barraquer in his cataract work has one complete set of instruments freshly sterilized for each patient to be operated on. Each set is arranged in a neat compact metal box with removable cover. This box with its set of instruments is enclosed loosely in a paper envelope. In this envelope it has been previously sterilized by dry heat, and the seal is not broken until everything is in readiness for the operation to proceed. Then the operating room nurse tears off the end of the envelope and slides the metal box out on the tray without contamination. The assistant removes the cover and arranges the instruments always in the same order.

The set contains—1 Panas cataract knife with double cutting edge.
1 Demarres lid elevator
1 Barraquer iris forceps

1 Barraquer iris scissors
I Iris spatula
1 Landolt fixation forceps
1 Needle holder without lock

Needles and thread are sterilized separately and are in another container.

The cleansing and disinfection of the eyelids and surrounding parts having been done in preparation for the operation, the instillation of the mydriatic and anæsthetic begins one hour previous to operation. For this purpose a combination ointment of cocaine and euphthalmine is used. Instillations of this ointment into the inferior conjunctival sac are made every ten minutes for one hour, and the patient then comes to the operating table. On the table the patient is given an hypodermic of novocaine along those branches of the facial nerve so as to paralyze, temporarily, the action of the orbicularis according to the method of von Lindt. The face is then covered with sterile gauze leaving both eyes exposed. Both operator and assistant now put on sterile white cotton gloves.

The assistant is seated at Dr. Barraquer's right, and raises the upper eyelid away from the eyeball with the Demarres lid elevator. The lower lid is held away from the eyeball by the thumb of the assistant's other hand. With Landolt forceps the operator causes fixation without pressure by grasping the conjunctiva and sclera about *2mm* from the limbus at its lower part. With the cataract knife the puncture and counter-puncture are made so as to include slightly less than one half the circumference of the cornea. Particular attention is given to the making of the incision so that its under surface will give the largest possible smooth aperture, and the upper third is made with a conjunctival flap. This conjunctival flap is next turned down over the cornea and the Barraquer iris forceps and scissors are introduced at the tip of the wound above, and the smallest possible peripheral iridectomy is made without bringing the iris out of the eye. The two conjunctival sutures are next placed and one twist of the knot of each is made. These sutures are put out of the way one on each side of the eyeball.

The motor of the suction pump is now started and the

operator takes the erisiphake in his hand very much as one would hold a pen, the ball of the thumb operating the valve. With the suction constantly at the desired level and the valve closed, the tip of the erisiphake is introduced into the anterior chamber, and without touching the iris, the tip is placed a little beyond the center of the lens and the valve opened. At the instant the valve is opened it will be noticed that the lens is gripped in the tip of the ventouse. Immediately the operator starts to roll the lens out of the patellar fossa by a steady rotating motion without producing the slighest pressure on the vitreous. The lens is thus removed from the eye upside down as it were. The small peripheral iridectomy has allowed the air to follow in the wake of the extracted lens, and so no suction or vacuum is produced in the patellar fossa, which is an important factor. The conjunctival sutures are now tied and, if necessary, stroking of the lips of the wound is done to place everything in proper apposition. Manipulation of the iris is rarely necessary, but should the pupil be out of position or uneven, the iris is gently stroked into place, the spatula being inserted into the wound along the side.

Eserine ointment is now instilled into the conjunctival sac and a light pressure bandage applied to both eyes. The patient is allowed to sit up, step down from the table, and walk to his bed. If the patient has been operated with his clothes on, and many of them were operated fully clothed, he is expected to remove his clothes within the next two hours and go to bed.

In those cases where everything was perfect and the operation passed without incident, the original bandage would not be disturbed for five days, although the patient would be allowed up out of bed before that time. Others would be examined sooner according to the exigencies of the case.

On the fifth day the bandage is removed sufficiently to examine the eye to note the reformation of the anterior chamber, the position of the iris, and the general appearance of the eye. It was astonishing how free from redness these eyes would be, and also the clearness of vision of the operated eye on its first examination.

Since I returned I wrote Dr. Barraquer asking for statistics of the cases operated while I was in Barcelona and he was kind

enough to send them to me. Arranged in percentages they are as follows:

25%	had vision	=	1.	or $\frac{20}{20}$
10%	" "	=	.9	" $\frac{20}{22}$
20%	" "	=	.8	" $\frac{20}{25}$
15%	" "	=	.6	" $\frac{20}{35}$
10%	" "	=	.5	" $\frac{20}{40}$
5%	" "	=	.4	" $\frac{20}{50}$
5%	" "	=	.3	" $\frac{20}{65}$
10%	" "	=	.2	" $\frac{20}{100}$

These tests were made mostly on the 15th day following the operation. Thus on the 15th day 52% had a vision of .8 or better. 76% on the 15th day had a vision of .5 or better.

The complications that would arise are mostly due to a faulty technic. If the amount of vacuum used is too strong for that particular lens, a rupture of the capsule may occur. If it occurs as soon as the vacuum is applied and while the lens is in its patellar fossa, it may be impossible to lift the lens from the eye and in that case the operator should cease working with the erisiphake and remove the lens by the usual expression method. If the rupture occurs as the lens is near its exit from the eyeball, the operation should proceed and the lens débris be stroked from the anterior chamber before the sutures are tied. If the vacuum is insufficient to cause the tip of the ventouse to adhere to the lens, or if the tip is improperly placed, a sucking noise will be instantly heard when the vacuum is applied. The valve should then be immediately closed. If it is ascertained that the tip is improperly applied, a correction should be made and another attempt made at removal. Should the vacuum be insufficient an adjustment of the vacuum by the regulator may be made and the operation proceed.

The presentation of vitreous would be an indication of pressure on the eyeball, unless it would be one of those rare cases of choroidal hemorrhage induced in sclerosed vessels through the sudden reduction of intraocular pressure. Such an hemorrhage occurred in one case operated by Dr. Barraquer while he was in America and I was told an enucleation became necessary. I believe such an hemorrhage would have

occurred no matter what method of extraction had been used as the expulsive hemorrhage occurred before any attempt had been made to introduce the erisiphake.

The main object of this operation is to remove the cataractous lens in its capsule without exerting pressure on the vitreous body so as not to cause a rupture of the hyaloid membrane, as occurs so frequently in the Smith-Indian method. Not one of the cases that had been operated by phacœrisis, that I examined with the binocular microscope while I was in Barcelona presented an hernia of the vitreous. One patient I examined had had a cataract operation on each eye, one eye by the Smith-Indian method and the other by phacœrisis. In the eye operated by the Smith-Indian method there was vitreous hernia, but it was absent in the eye on which the phacœrisis method had been used. When the vitreous presents it should be treated in the same manner as with any other cataract operation. Iritis is less frequent than where the capsulotomy method is used. Dr. Barraquer says that iritis is rarely present if the operation proceeds without incident. A striped keratitis may be present at the first dressing, but it is usually insignificant and disappears without any special treatment before the patient is ready to leave the hospital. This striped keratitis is probably induced by the erisiphake coming in contact with the endothelial cells of the cornea.

Complications following the phacœrisis method are less than by the Smith-Indian or capsulotomy methods of extraction. It is an operation requiring great dexterity, but not more difficult than the expression method.

Prof. Gallemaerts of Brussels says, the erisiphake of Barraquer does away with the pressure on the vitreous body which is so brutal in the operation of Smith. The results give great satisfaction, no irritation, a black pupil, excellent vision, and above all, no secondary cataract to be reckoned with.

In closing I would say to all those intending to adopt the operation of Barraquer, as Prof. Gallemaerts has said, "one should not attempt to do the operation without first having made a pilgrimage to Barcelona."

A DERMOID OF THE CORNEA.

By Dr. T. HARRISON BUTLER, Birmingham, England.

(With one illustration on Text-Plate XXXIII.)

THE general appearance of the condition to be described is shown by the drawing which was made by my House Surgeon, Mr. G. B. Lowe. The Case History is as follows:

Miss D. F., aged 19, came to the Birmingham Eye Hospital on February 14, 1922, complaining that something was growing upon her left eye. It had been noticed for a month, and had rapidly reached its present size. There had never been any redness of the eye, and no pain.

The patient is a decidedly good-looking healthy brunette well under the average in height and weight.

On the outer aspect of the cornea, and somewhat invading the sclera there is a flat, yellowish white, somewhat raised, patch. It is about *6mm* in diameter, and is very slightly injected on its scleral aspect.

The vision of each eye was $\frac{6}{6}$ths, and the fundi were normal.

The general aspect of the growth suggested a dermoid cyst, but the short history was strongly against this view. I interviewed the mother and brother and both were certain that the eye was originally normal in appearance, and that the growth was of recent development. It was inherently improbable that a young good-looking girl would have an unsightly excrescence upon her eye and not be aware of it.

I had about a year previously seen a similar condition in another young girl, which, commencing like a large phlyctenule, began to invade the cornea. This was undoubtedly a malignant growth, and was completely cured with the maintenance of full vision by treatment with radium at the Radium Institute in London.

I got my colleague, Mr. Jameson Evans, to see the case, and we concluded that the most likely diagnosis was malignant growth of the cornea.

On Feb. 27, 1922, Miss F. went to London to the Radium

Institute with the object of having the growth irradiated. The authorities there, however, had some doubt about the true nature of the case, and they asked their ophthalmologist Mr. Holmes Spicer to see the case. He formed the definite opinion that it was a congenital dermoid cyst, and she was sent back to me.

On April 4, 1922, I showed the case to a Joint Meeting of the Midland and the South Western Ophthalmological Societies. She was examined by a large number of well-known Ophthalmic Surgeons, including Mr. Richardson Cross, and her case was fully discussed. Three diagnoses were considered: it might be malignant; it might be inflammatory, perhaps tuberculous; and finally it could be a dermoid.

The general opinion was that it was not malignant. Some inclined to the inflammatory theory, but the majority held that it was a degenerating dermoid.

April 25, 1922. The growth is now decidedly larger and begins to encroach upon the pupil area. It is more yellow, and the line of injection is invading the tumor instead of being confined to its edge. The eye itself is showing a general ciliary blush.

The patient was now admitted to Hospital and tested by injections of old tuberculine. Three injections were given at intervals of a week as follows: 0.001*mg* 0.002*mg* 0.004*mg*. There was no reaction of any sort. A short course of injections of T.R. had no influence upon the progress of the case.

The Wassermann was negative.

I took a thin shaving from the surface of the tumor with a Beer's knife. This was examined by Mr. Eric Assinder, Pathologist to the Hospital and he reported that it consisted of normal corneal tissue, but that the section was too thin for an accurate examination.

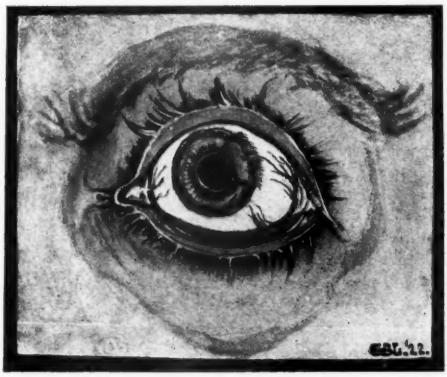
The tumor remained angry looking and showed no sign of yielding to treatment so I eventually persuaded the Radium Institute to irradiate the growth. This was done, but there was no obvious result, in fact for some time the eye was very irritable and injected.

In August the injection was less and at the end of 1922 the eye was free from all inflammation.

At the present time, May, 1923, the eye is perfectly white. The tumor has become flat and is yellowish white. It does not invade the pupil area, and the vision is still $\frac{6}{9}$ths.

The diagnosis is still open to doubt, but it seems obvious that malignancy can be definitely excluded. The most probable explanation is that there was at the limbus a small dermoid cyst that had not been noticed. This commenced to enlarge and then to degenerate with inflammatory reaction.

ILLUSTRATING DR. T. HARRISON BUTLER'S ARTICLE ON "A DERMOID OF THE CORNEA."



REPORT OF A CASE OF PURPURA HEMORRHAGICA.[1]

By Dr. MARTIN COHEN, New York City.

(With three illustrations on Text-Plate XXXIV.)

PURPURA hemorrhagica, first described by Werlhof in 1775, and also termed morbus maculosus Werlhofii though usually defined as a severe form of purpura, is in reality a definite clinical entity and should be designated as a primary idiopathic disease of the blood. In addition to the purple patches upon the skin and mucous membranes, due to subcutaneous extravasation of blood, which are characteristic of the more common form of purpura, this affection is attended by severe hemorrhages from the mucous membranes and many grave constitutional symptoms, the blood changes being especially marked.

There have been frequent reports of conjunctival and retinal hemorrhages occurring in the course of purpura hemorrhagica but I have been able to find only three such reports which contain microscopical eye findings, and even these are without the comparison or corroboration of hematological study. The first of these cases is that reported by Ruc in 1870; the second that of Pagenstecher, published in 1905; and the third, that observed by Marx the following year (1906). It seems, therefore, that a report of the following case will be of interest, especially in regard to the microscopical eye findings and the results of hematologic study:

J. S., male, married, born in the United States, aged 42, was admitted to Dr. Bullowa's service at the Harlem Hospital, November 18, 1921, because of subcutaneous

[1] Read before the Section on Ophthalmology, N.Y. Academy of Medicine, February 19, 1923.

hemorrhages over the surface of the body, extravasation from the gums, and general debility. The family history was irrelevant; the wife was in good health and had had no miscarriages; there were no children. No history of diseases of childhood was given, but the patient had had yellow fever in 1898. There had been excessive use of alcohol up to within six months of the time of admission.

The illness for which treatment was sought had begun three months before, ushered in by general weakness and lassitude. About two months before, blood had begun to ooze from the gums, and this had continued up to the time of admission, although the gums were never tender nor painful.

On inspection, the patient appeared much exsanguinated and asthenic; he was emaciated and very drowsy. Blood was oozing from the gums, the tongue was dry and coated with blood clots, and the breath of very foul odor. The teeth were in poor condition, pressure upon the gums causing pus to exude from the alveolar pockets, which on culture showed streptococcus viridans. Epistaxis was also observed.

The heart was not hypertrophied; the action was regular; there were no murmurs and the heart sounds were clear and distinct. The pulse, though thready and almost impalpable, was rapid and regular. The reflexes were normal; there were no glandular enlargements; and the lungs, abdomen, and rectum were negative. The Wassermann reaction was negative; no pus, blood, nor evidence of arsenic could be demonstrated in the urine, and the stools were negative. Blood pressure: systolic, 130; diastolic 80. Temperature, 99. There was no clinical evidence of arteriosclerosis.

The skin of the entire body showed hemorrhagic spots of various sizes scattered over the arms, chest, abdomen, flanks, thighs and legs, in many places becoming confluent. These spots did not disappear on pressure. The patient complained of poor vision in both eyes.

The pupillary reactions were normal; the field was normal for form and colors. The tension, musculature, anterior chamber, and iris were normal. The fundus examination showed symmetrical changes in each fundus, these changes having the appearance characteristic of old and recent hemorrhages. The disk margins were somewhat blurred but not elevated. The retinal vessels showed no alterations except venous hyperæmia. An old circular hemorrhage and several recent linear ones were observable in the circumpapillary area, some of them showing the central yellowish dot characteristic of blood changes in general. In the macular area was a pre-retinal hemorrhage which appeared to be older than those previously mentioned.

ILLUSTRATING DR. MARTIN COHEN'S ARTICLE ON "REPORT OF A CASE OF PURPURA HEMORRHAGICA."

FIG. 1.

Coagulation of red blood corpuscles and fibrin in sinus angle and spaces of fontana.

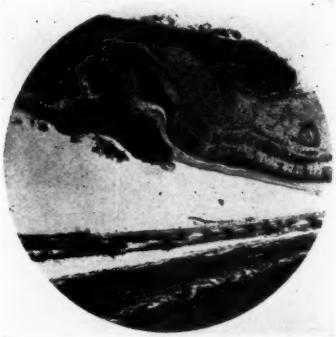


FIG. 2.

Pre-retinal, inter-retinal, and sub-retinal hemorrhage.

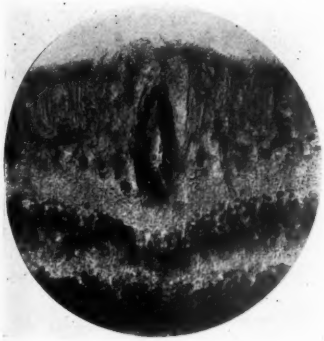


FIG. 3.

Medial thickening of artery.

In the periphery, three large ill-defined foci were scattered in the retina, dull white in color and not surrounded by pigment nor blood. Two of these foci were absorbed later.

Retinal hemorrhages are of the same nature as those taking place in other parts of the body in cases of purpura hemorrhagica, and are believed to be due to a marked diapedesis of red blood cells brought about by the pathological condition of the blood. The blurring of the disk margin and the dull white foci were probably caused by edema, secondary to a venous stasis. The yellowish dots seen in the center of the hemorrhagic areas are explained as collections of leucocytes.

The hematological examination on admission showed:

Red cells,	650,000	Hemoglobin,	15%
White cells,	9,000	Polymorphonuclears,	80%
Platelets,	82,000	Lymphocytes,	20%

Bleeding time, 9 minutes
Coagulation time, 18 minutes

Fragility test of red blood cells, normal. As noted above, the blood platelets were markedly diminished.

The clinical diagnosis was purpura hemorrhagica.

The treatment consisted of intravenous injections of calcium chloride and sodium citrate, and several transfusions of whole blood. Splenectomy and deep X-ray treatment were also suggested, but were not tried.

The patient died from exsanguination on December 23, 1923, thirty-seven days after admission. At autopsy, nothing pathological was observed macroscopically. Unfortunately, no microscopical sections were made for the study of a diffuse vascular disease. An hour after death, one eye was removed and fixed in 10 per cent. formalin. After fixation the eye was embedded in celloidin, and then sectioned and stained with a modified van Gieson. Forty-five sections of the eye were studied, although no serial sections were made.

Microscopical Examination.—The cornea was normal. The anterior chamber showed a coagulation of fibrin and red blood corpuscles located in the sinus angle and also red blood cells in the spaces of Fontana (Fig. 1). The uveal tract and vitreous were normal.

Retina.—A large pre-retinal hemorrhage was seen in the peri-macular area, and near the papilla was a sub-retinal hemorrhage (Fig. 2), also all the layers of the retina were suffused with red blood corpuscles,—indicating hemorrhages oozing mainly from capillaries,—while the larger vessels

were not surrounded with hemorrhages. The smaller vessels showed a moderate medial thickening of their walls, mainly of the arteries (Fig. 3). Some of the vessel walls were permeated with a few red cells. The endothelial lining was apparently normal.

The choroid showed a slight medial thickening of the vessel walls, but was otherwise normal. The papilla was moderately swollen.

The characteristic microscopical eye findings were: First, the hemorrhages; second, the moderate medial thickening of the vascular walls of the smaller arteries and veins. The hemorrhages were due to diapedesis of the red blood corpuscles, probably dependent on an unknown pathological condition of the blood. The medial wall thickening was either part of an early arteriosclerosis; or it was secondary to a changed condition of the blood; or the thickening occurred simultaneously with the retinal hemorrhages, the hemorrhages appearing mainly at areas some distance from the vascular wall thickening.

Most authorities are of the opinion that vascular wall lesions are not a factor in this disease. In this case, however, it would seem that the vascular wall thickening noted in the retina and choroid was part of the general disease, although there was no clinical evidence of an arteriosclerosis. It is not usual to find the retinal and choroidal vessels thickened to such an extent in an individual the age of this patient. Unfortunately, no microscopical examination was made of the vascular structures elsewhere in the body.

In conclusion, I wish to call attention to the necessity of microscopical eye examination in this rare disease, on account of the few reports in the literature, and also of the possibility of increasing our knowledge of the pathological alterations of the vessel walls in this disease by studying the vascular changes in the retina and choroid.

SUBPERIOSTEAL ABSCESS IN THE FLOOR OF THE ORBIT.[1]

By Dr. ARNOLD KNAPP, New York.

IT is stated that of the accessory nasal sinus inflammations, maxillary sinusitis is second to the frontal sinusitis in frequency as a cause for orbital infections. Birch-Hirschfeld[2] says that in suppuration of the maxillary antrum there is a high percentage of blindness; of 89 cases, transitory visual disturbances occurred in 11, blindness in 24, and death in 13, possibly because orbital abscess is particularly frequent after maxillary suppuration. An explanation is found in the communication of the veins of the antrum with the inferior ophthalmic vein. A subperiosteal abscess, or a periostitis secondary to an antral infection, is situated in the lower part of the orbit, with swelling of lids, chemosis, exophthalmos, displacement of the eyeball upward, and restricted motility downward as characteristic symptoms. A fistula sometimes develops at the lower orbital margin. In view of the frequency of maxillary infection, it is strange that a periostitis at the floor of the orbit is not more often observed. I have been on a particular lookout for this localization of an orbital periostitis, because I could never explain why the upper wall of the antrum should from its anatomical position serve so often as an intermediary for a pyogenic process.

The following case was therefore of the greatest interest to me:

B. B., aged 29, was seen on February 27, 1923, stating that after an attack of influenza three weeks ago, the left eye had begun to protrude one week later. There was left ptosis, the eye was pushed forward and up and partly

[1] Case presented before the Ophthalmological Section, New York Academy of Medicine, April 16, 1923.

[2] *Graefe-Saemisch Handbuch*, II. ed.

immobile. The lids were normal; conjunctiva congested; pupil moderately dilated; optic disk normal. An incision had been made in the lower fornix, which contained a gauze drain. On removing there was no discharge. Along the entire floor of the orbit there was a uniform thickening. The nose was examined by Dr. H. P. Howell, who found some pus in the left middle meatus; the antrum was washed out, and contained pus. A wet dressing was applied. February 28: no fever. Swelling in floor of orbit seemed less, the eye about the same. Vision $\frac{1}{1}\frac{0}{5}$. The antrum was again washed out. March 2: the condition of the orbit was about the same. Point of greatest tenderness was on the floor, just outside of the lacrimal groove. As the condition was not improving, and fearing the development of an orbital abscess, it was decided to operate.

Operation.—Incision along the lower orbital margin; the periosteum was detached from the underlying bone. At about *2cm* posterior to the bony margin there was a defect in the bone leading into a cavity partly in the floor and in the adjoining inner wall. Some granulations were removed. There was no free pus. Gauze packing. The cutaneous wound was left open. Opening into the antrum from the nose was enlarged. March 4: packing removed. There was retention of purulent fluid, and a rubber tube was introduced. The discharge from the nose had increased. March 5: considerable purulent discharge through the tube. Irrigating the antrum, or blowing the nose, caused a discharge to appear in the orbital wound. Swelling of the eye was distinctly less. No fever. March 12: the antrum had cleared up. Secretion from the tube lessened, and the tube was left off. Gauze packing introduced. The swelling of the lids and the exophthalmos much less. The inferior rectus still remained weak. March 17: returned home. The wound had been dry for several days. No packing. Nose clean. Exophthalmos was very slight. Motility downward was still restricted. April 15: no external deformity. Slight œdema of lid. Motility normal. Interior normal. Vision normal.

The location of the bone defect found at operation proved that this was an empyema of an ethmoidal cell which perforated both into the orbit and in the antrum. This ethmoidal cell belonged to that group which extend outward in the roof of the antrum. The perforation into the orbit causes thickening of the periosteum at the floor of the orbit, just external to the lacrimal groove.

The case was therefore not primarily an antral infection but that of an ethmoidal cell, a condition which much more easily

explains a periostitis at the orbital floor than an isolated antral suppuration.

The method of operating, consisting in a cutaneous incision along the lower margin of the orbit, with retraction of the periosteum, permits exploration of the bony wall and definite localization of the diseased area.

REPORT OF THE BRITISH MINISTRY OF HEALTH ON THE CAUSES AND PREVENTION OF BLINDNESS.

By Dr. P. G. DOYNE, London.

THIS committee was appointed by the minister of health—Dr. Addison—in September, 1920, "to investigate and report on the causes of blindness including defective vision sufficient to impair economic efficiency and to suggest measures which might be taken to prevent blindness."

Most of the information was obtained in England and Wales and to a lesser extent in Scotland. The register of the blind for England and Wales, March, 1921, shows 34,894 persons registered as blind. The definition of blindness adopted was "too blind to perform work for which eyesight is essential."

For the purpose of the investigation the following material was obtained: with regard to infants, figures were supplied by Mr. Bishop Harman and by the Board of Education from the medical officers of the various schools; Mr. Bishop Harman in addition submitted evidence from his examination over the past 17 years of 3300 blind and partially sighted children and also from an analysis of 601 cases of blindness of all ages. Further, examination was made of blind workers in London, Liverpool and Bristol by Mr. P. G. Doyne, Mr. H. R. Bickerton and Dr. A. Fells respectively. Mr. Doyne also examined the blind residents of the North London Home for the blind of whom all but 5 were over 50 years of age. To establish uniformity as far as possible, a uniform scheme was adopted in the examination of the blind workers of London, Bristol and Liverpool. The age of the onset of blindness was as far as possible ascertained in the following age groupings, 0–5, 6–15, 16–50, and over 50. The report points out the extreme

difficulty in some cases, where the blind eyes were old and shrunken or where the eyes had been previously removed, in determining the exact nature of the initial lesion. In a brief summary the report points out that in infancy ophthalmia neonatorum is the prime cause of blindness, various congenital defects coming next in frequency and that important factors were interstitial keratitis, diseases of the posterior parts of the globe and to a lesser extent myopia.

In middle life, accidents assume a high proportion and iritis and iridocyclitis advance relatively. After middle life glaucoma assumes a high position and cataract appears as a cause of blindness (only cases where operation had been unsuccessful were included), the other factors prominent in earlier life showing a proportionate decrease. The report then proceeds to deal with certain causative factors in greater detail.

OPHTHALMIA NEONATORUM.

Statistics show that this disease is a serious factor in the causation of blindness, the individual being blinded from birth and throughout the years of highest economic efficiency. A lessened proportionate incidence in later years is shown and this is due to the death of affected persons and the emergence of other factors. Ophthalmia neonatorum has been made notifiable throughout England and Wales since 1914. It is defined "as a purulent discharge from the eyes of an infant commencing within 21 days from the date of birth." Notification from England and Wales showed roughly that the incidence of the disease was 10 per 1000 births. But statistics from various counties and boroughs varied widely. This was largely due to differences in the details of the notification in different areas. Further the duty of notification was laid upon the certified midwife attending the confinement unless the case had already been notified by a medical practitioner. In certain areas only those cases, notified by the midwife, but which had been verified by the doctor, were considered.

The report points out that the primary object of notification was to ensure early and complete treatment and that this rather than accurate statistical records was the object in view and in consequence the widest possible classification for noti-

fication was desirable. On the whole since 1914 the incidence of the disease does not appear to have diminished, but against this there is the most definite statistical evidence that the percentage of impairment of vision and blindness due to this cause has been greatly reduced.

The report urges early treatment at the outset and after care of cases in whom the active stage of the disease has been overcome as in these cases the final visual result may not be apparent at first. Ophthalmia neonatorum is not necessarily venereal in origin but at least 50 per cent. of the cases are of this nature and these are usually the severe cases. Consequently the prevention of O. N. is part of the wider question of the prevention and treatment of venereal disease. With regard to prevention, there is firstly the prevention and treatment of venereal disease in general; the next line of defence is the treatment of the expectant mother—anti-natal treatment is strongly stressed in the report—; then prophylaxis at birth. This latter is largely in the hands of the midwife and can be influenced by the proper training of midwives and by the imposition of suitable punishments in cases of proved carelessness. The report urges that more instruction should be given to midwives concerning this disease and that they should be made to visit ophthalmic hospitals for the purpose of gaining direct experience of O. N. The report does not consider that Crede's method—"2 per cent. silver nitrate drops instilled within the lids immediately after birth"—should be used as a routine by midwives. The excellence of this method is acknowledged but it was thought that an undue sense of security so produced by its use might militate against the scrupulous cleansing of the eyes which is even more important and also that the "inflammatory reaction" often produced by the method might either cause the midwife to notify the condition as O. N. or on the other hand she might mistake the actual onset of O. N. for the "inflammatory reaction." Crede's method should be reserved for the medical man. The report considers that even at the present time cases of O. N. are often not treated as soon as should be and urge that facilities for consultation with an ophthalmic surgeon should be provided wherever practicable. The report recommends that the ministry of health should take steps to insure that adequate nursing

provision is always available for cases of O. N. which are not admitted to hospital. The report also recommends that the general medical council be urged to insist that every student presenting himself for the medical qualification examination shall be examined in ophthalmology. The report urges the advantages of hospital accommodation for the mother along with the infant who is being treated, so that she may continue to feed her infant and may herself be treated if it is necessary.

SYPHILIS.

From the information supplied it was estimated that syphilis was the direct cause of from 10 to 15 per cent. of the blindness existing in the United Kingdom. Attention is drawn to the fact that it is the innocent that suffer most, as evidenced by the high incidence of interstitial keratitis. Syphilitic blindness is but a part of the whole problem of syphilis and its diminution will be dependent upon the success attending anti-specific measures of treatment and prevention.

CONGENITAL MALFORMATION OF THE EYES.

The report states that between 20 and 30 per cent. of blindness in children is due to congenital malformations. But many of these conditions are amenable to treatment which in successful cases will produce a degree of useful sight. The report notes that there is as yet not sufficient knowledge available as to the ætiology of these conditions by which preventive measures can be adopted. An inherited tendency is often demonstrable and where this is present prevention would appear to lie in eugenic measures with regard to marriage.

SURFACE INFLAMMATIONS OF THE EYES IN CHILDHOOD APART FROM OPHTHALMIA NEONATORUM AND TRACHOMA.

Under this heading are included purulent conjunctivitis due to measles, phlyctenular disease and blepharo-conjunctivitis. These conditions are not very common causes of blindness but disable children for long periods from school and are character-

ized by great chronicity and a great tendency to relapses and also finally to considerable impairment of sight. Poverty and bad hygienic conditions are the real causes of the diseases and the report urges for further powers for the local authorities so that children suffering in this way may be treated in special schools if possible in the country.

TRACHOMA.

This disease is now a very small factor as a cause of blindness. Statistics show a marked decline in the number of entrances to the special trachoma schools. It is now in this country chiefly due to introduction from abroad.

MYOPIA.

This is a cause of a quite considerable amount of blindness and a still further amount of serious impairment of sight. Attention is drawn to school myopia and the special myope classes, instituted by the London county council, are fully approved. The importance of occupation for the myope is stressed as so many of these cases suffer failure of sight, by reason of unsuitable employment, in early middle life. Some very striking statistics are produced with regard to this point. In a series of 480 myopes of over 3 diopters (183 of whom were engaged in habitual close work, 297 of whom were not so engaged); of the 183 habitual close workers 97—53 per cent.—broke down on account of failure of sight, whereas of the 297 who were not engaged in close work 28—9.4 per cent.—broke down from eye failure.

GLAUCOMA.

This disease becomes a serious factor in the production of blindness after middle life. The determining cause of glaucoma is as yet unknown. In consequence it cannot be definitely included among the preventable diseases. The report draws attention to the importance of early diagnosis and treatment. The report draws attention to the danger of persons over middle age attending unqualified persons who will not recog-

nize the condition and will attempt to treat the condition by supplying glasses.

INDUSTRIAL EYE DISEASE AND ACCIDENT.

Industrial disease, apart from miners nystagmus, appears to be a small cause of blindness or impairment of sight. The amount of injury to vision caused by accident is however considerable. Statistics on this point are not fully available and the report recommends that the information possessed by insurance companies might well be pooled so that definite information should be available for government departments. A report from the Scottish Board of Health shows that out of 5515 blind persons of all ages on the Scottish register of the blind there were 426 (7.2 per cent.) whose blindness was due to accidents occurring in occupations.

Lead.—The evidence appears to show that blindness due to lead poisoning is small in amount and decreasing. The stringent measures enforced in industries concerned with lead are therefore apparently successful. Of these measures the most important are: (1) removal of lead dust by exhaust ventilation; (2) the use of leadless and low solubility glazes; (3) introduction of labor saving devices; (4) the periodical medical examination of workers.

Derivatives of Benzene and Carbon Bisulphide.—No case of optic neuritis has been definitely established as due to these causes.

Methyl Alcohol.—There appears to be no authenticated instance of injury to eyesight from this cause in the United Kingdom. The committee however draw attention to the danger which may follow upon its use.

Glassworkers and Metal Workers Cataract.—This form of cataract first appears as a post cortical cataract and later matures to a total cataract. In the early stage it causes little impairment of sight and later can be dealt with operatively. It is chiefly important as a cause of loss of working capacity.

Corneal Ulceration Due to Pitch.—Four certificates of this disablement were returned in 1920. Goggles to give protection is recommended as the surest protection.

Miners Nystagmus. This is the most serious industrial

disease. The committee hold that deficient illumination is the essential factor. In 1920 there were 7028 cases in receipt of compensation of whom 2865 were fresh cases that year.

EYE ACCIDENTS IN FACTORIES AND WORKSHOPS.

Such accidents are at the present time notifiable. The proportion of eye accidents to other accidents averages about 4.5 per cent. On this basis approximately 6000 industrial eye accidents occur per annum. No reliable information is obtainable as to the resultant visual loss as the notification is made at the time of the accident. It is chiefly in the metal and engineering trades that eye accidents are most frequent. Flying pieces of metal, splashes of molten metal and small fragments from lathes and drilling machines are the principal agents of damage. The committee made special inquiry into three branches of industry in which there was a high eye injury incidence.

Aerated Water Manufacture.—Although there are not a large number of persons employed in this industry yet the eye accidents are proportionately great. The last reliable statistics are those of 1903. These show that this trade contributed 10 per cent. of all industrial eye accidents. Since then however there has been a great improvement in the machinery employed and it is hoped that the danger in the filling process is now eliminated.

Textile Industries.—Here the damage is caused by the flying shuttle. The incidence of damage is very small, but the damage itself is usually of great severity. The committee advise that further investigation is desirable in order to improve the guards to the shuttles.

Industries Involving the Use of Chemicals.—Here in spite of the great possibilities for damage to the eyes, the actual figures are most satisfactory. The committee were very favorably impressed with the efforts made to secure protection and treatment of such accidents as occur. Goggles and protective masks were the most successful measures together with screens and metal guards to the pumps. Different types of goggles are needed for different processes. It was sometimes difficult to get the men to wear goggles.

CONSIDERATIONS WITH REGARD TO PREVENTION.

The factory act requires that the certifying surgeon shall give a certificate to every child or young person under the age of 16. There is therefore some initial control, but there is no further control of defects of vision in persons who enter industry over the age of 16. The committee consider the question of accurate vision as most important though admitting that there are many processes in the working of which full standard vision is not necessary. They point out the need for correcting glasses and hold that it is desirable that persons in industry over the age of 16 should be subjected to eye tests. With regard to lighting it has been shown that the accident rate as between artificial and natural lighting is far higher in the former even to the extent of 40 per cent. in some cases. Excess of light may be as harmful as deficiency of light. Except in a few special processes, there are no legal requirements affecting the lighting of factories and workshops. Improvement in mechanical devices is the most effective way of preventing accidents. An extension of the use of screens on lathes and abrasive wheels and of the use of side screens in order to give protection against flying particles is urged. Localized exhaust ventilation is valuable as a protection against both dust and small foreign bodies.

THE WEARING OF PROTECTIVE GOGGLES.

It would appear obvious that considerable increase in safety could be attained by the wholesale wearing of protective goggles. There are however many difficulties in the way. There is the natural prejudice on the part of the men, particularly among the older men who consider the habit effeminate and derogatory as employing lack of skill. There are certain other disadvantages consisting in the curtailment of the visual field, discomfort in wearing, steaming of the glass, etc., which tend to reduce working capacity. The committee considers that in this country insufficient scientific attention has been paid to this construction of such protective goggles. They lay down certain general requirements:

1. All injurious particles should be prevented from reaching the eyes from in front, from either side and from below.

2. The goggles should be light and allow a free play of air so that no condensation of moisture on the transparent medium can occur.

3. They should not impede vision.

4. They should not be liable to become obscured by the impact of particles.

Further the committee recommend that steps should be taken to issue detailed specifications of the most suitable goggles and masks for the differing industrial processes.

The importance of promptitude in securing treatment of an eye accident is strongly emphasized by the committee.

EYE ACCIDENTS IN MINES AND QUARRIES.

Statistics furnished by the Scottish Board of Trade show that out of 427 blind persons whose blindness was directly due to industrial accident there were 103 miners and 26 quarrymen. The accidents are mostly due to flying fragments of mineral or metal. With regard to mines the committee think that the wearing of goggles is impracticable and would consider that improved illumination was the most certain preventative. With regard to quarries the extended use of goggles is advised.

EYE ACCIDENTS IN AGRICULTURE.

Injuries from thorns, branches, etc., and from threshing, dyking and loading corn cause the majority of the accidents. In tending cattle injuries may occur. The industry appears to be too little organized for effective means of control.

EYE ACCIDENTS IN CIVIL LIFE.

A great proportion of these occur before the age of 16. Their prevention in so far as they are preventable appears to be a matter of individual common sense and caution.

SYMPATHETIC OPHTHALMIA.

Blindness due to this cause has largely decreased in later years and is due probably to improved technique in the early

treatment. The committee draws the attention of the general practitioner to the ever present danger of this disaster in all cases of penetrating wounds of the eye.

THE TREATMENT OF EYE DISEASE AND ERRORS OF REFRACTION BY UNQUALIFIED PERSONS.

The committee is of the opinion that it is not desirable and a danger to the public that a person who is qualified to make glasses prescribed by a medical man should be given any degree or qualification which might convey to the mind of the lay public that such a person was competent to treat eyes and prescribe glasses.

REPORT ON THE PROGRESS OF OPHTHALMOLOGY.

Abstracts by Drs. A. N. Alling, New Haven; M. J. Schoenberg, New York; T. H. Butler, London; P. G. Doyne, London; and K. Wessely (*Archiv für Augenheilkunde*), Würzburg.

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(*Continued from the July issue.*)

XII.—THE LENS.

86. Böhm, Ferd. **Extraction of the transparent lens in myopia.** *Archiv f. Ophthalmologie*, ciii., 2, 1920.

87. Green, A. S. and Green, L. D. **Vacuum method of intracapsular cataract extraction.** *American Journal of Ophthalmology*, February, 1922.

88. Jess. **Problem of the origin of cataract.** *Deutsche med. Wochenschr.*, 1921, p. 284.

89. McDannald, C. E. **Vacuum extraction of cataracts.** *American Journal of Ophthalmology*, February, 1922.

90. McReynolds, John O. **Professor Barraquer of Barcelona and his method of phakœrisis.** *Ibid.*, February, 1922.

91. Schneider, Curt. **Cataract operation with total and peripheral iridectomy.** *Inaug. Diss.*, Leipsic, 1920.

92. Smith, H. **Mature and immature senile cataract.** *American Journal of Ophthalmology*, December, 1921.

93. Zentmayer, W. **Results of cataract operations performed by Col. Henry Smith at Wills Hospital, Philadelphia, Pa.** *Ibid.*, February, 1922.

Smith (92, **Mature and immature senile cataract**) gives the history of the intracapsular extraction and compares its technic and results with the ordinary operation. Alling.

Zentmayer (93, **Results of operations performed by Col. Smith**) records the results obtained by Col. Smith in operating on 18 cataracts at Wills Hospital. He thinks that the free use

of 1–3,000 bichloride is undesirable and that the section causes unnecessary drag on the anterior parts of the eye. The vitreous loss in 38%, incarceration of iris 38%, secondary glaucoma in one. In most of the cases there was vitreous haze or definite vitreous opacities. ALLING.

MCREYNOLDS (90, **Professor Barraquer of Barcelona and his method of phakœrisis**) records his impressions received from a visit to Barcelona. The operation is not suitable except in patients over 50 years of age and is especially contraindicated in the case of dislocated lens and posterior synechiæ. He suggests that injury to the endothelium on Descemet's membrane might be the cause of complications and that the failure to apply the suction cup accurately to the anterior capsule might result in drawing the vitreous into the anterior chamber.

MCDANNALD (89, **vacuum extraction of cataracts**) has devised an instrument along the lines of the erisiphake of Barraquer but instead of employing a valve for control of the air pressure he has made a hole in the hollow handle over which the finger can be slipped to create suction at the tip. He believes that this method has possibilities when applied to selected cases.

GREEN (87, **Vacuum method of intracapsular cataract extraction**) calls attention to some of the dangers of this new method and has improved the instrument of Barraquer by using a foot valve for control of the suction thus relieving the tension on the hand while extraction is in progress. They regard as unsuitable cases those under 60 years of age, Morgagnian cataract and those in patients with prominent eyes. There is danger of rupturing the capsule by too strong suction and the pull required to rupture the zonular may result in detachment of the retina.

BÖHM (86, **Extraction of the transparent lens in myopia**) reports the results obtained in forty-five eyes operated on in thirty-four patients. Eighty-six per cent. were improved, 13.3% remained unchanged, 0.7% were made worse. The lowest degree of myopia was 16 D. No patient was under 10 years of age, or over 40. Serious general disease and retinal detachment, or practical blindness, of the other eye served as contraindications as long as the patient was able to work. Only one eye was operated on at first, the other not less than

two years after a satisfactory recovery. When possible only one eye was operated on, this one to serve for distant vision while the other was left unchanged for near work. The operation employed was discission and extraction some days later, leaving a round pupil. Secondary discission was needed in about half the cases; it never had to be repeated. Healing took place in from ten to twenty-one days, rarely required longer. Prolapse of iris occurred once, without influence on the result. Loss of vitreous occurred three times, glaucoma once, which was overcome. No iritis or signs of infection. Detachment of the retina did not occur early, but once occurred late.

JESS (88, **Origin of cataract**) attempts to explain the process of cataract as a hydrolytic breaking down of the albumin of the lens by ferments, and says that a knowledge of the exact composition of the proteins in the lens is necessary to further advance in this study.

SCHNEIDER (91, **Cataract operations with total and peripheral iridectomy**) compares these two procedures from the standpoints of the final vision, the degree of the final astigmatism, the frequency of prolapse of the iris, the frequency of loss of vitreous, the frequency of infection, and the frequency with which operation for after cataract is needed. From each of these viewpoints peripheral iridectomy appears to be preferable. The vision depends materially upon the permanent astigmatism, and he finds high degrees of corneal astigmatism more common after total than after peripheral iridectomy. Prolapse of the iris occurred in 3.72% of the cases in which total iridectomy had been performed, in 2.48% of those with peripheral. Loss of vitreous occurred in 5.84% with total, in 1.59% with peripheral iridectomy. Other conditions influence the figures regarding infection, but those given are: 3.01% with total, 1.95% with peripheral iridectomy. The figures concerning after cataract operations are not given; they are said to be difficult to compare, but to favor peripheral iridectomy.

XIII.—GLAUCOMA.

94. BRANDT. **Trephining.** *Archiv f. Ophthalmologie*, ciii., p. 95.

95. GIFFORD, H. **Peripheral iridotomy (Curran) in the treatment of glaucoma.** *American Journal of Ophthalmology*, December, 1921.

96. HERTEL and CITRON. **The osmotic pressure of the blood in patients with glaucoma.** *Arch. f. Ophthalmologie*, civ., p. 149.

97. KOYANAGI. **Glaucoma and cataract after wasp sting.** *Klin. Monatsbl. f. Augenheilkunde*, lxv., p. 854.

98. SEIDEL. **Permeability an essential property of the scleral scar after successful trephining.** *Arch. f. Ophthamologie*, civ., p. 158.

BRANDT (94, **Trephining**) has collated the results of 643 operations on 408 patients at the Heidelberg clinic. Cases of simple glaucoma numbered 146 eyes and 177 trephinings. His classification of results seems a little peculiar. Good results are those in which the vision is at least not materially impaired and the tension remains high; satisfactory those in which the tension became normal but the vision fell to a certain degree, while all the rest are included as bad. The longer the eyes remain under observation, the poorer the results. Of the eyes under observation for a year 78.8% showed good results, 9.2% medium, and 12.1% bad, while 90% of those observed less than three months showed good results. Most of the eyes were trephined only once, sixteen twice, five three times, and one eye five times. Of twenty-two eyes that had been operated on otherwise, sixteen were cured by trephining. Eighteen simple glaucomas were treated with iridectomy, for the most part with bad result. The worse the visual field before trephining, the sooner further contraction took place. The improvement of vision varied. In inflammatory glaucoma the results were worse than in simple, good in 72.5% of forty-one eyes. In many cases trephining was performed only after iridectomy had failed. Twenty-five cases immediately trephined gave 68% good, 8% satisfactory, and 40% bad results, while fifty cases treated with iridectomy gave 48% good, 6% satisfactory, and 40% bad results. To protect from degeneration unirritated eyes blinded by glaucoma 55 trephinings were performed on 49 eyes; good tension was secured in 68%. In irritated eyes of the same class the results were not so good; iridectomy was usually performed with 35% good results. In secondary increase of tension the operation was successful in half of the cases. Buphthalmos in children responded to trephining, as to sclerotomy, in the proportion of 3 cured to 4 not cured. The operation was usually performed under local anæsthesia produced by six drops of a 10% solution of cocaine with adrenalin.

The place of trephining was first upward and outward, then upward and inward, downward and outward, downward and inward. The trephine first pointed toward the midpoint of the eye, then its direction was slightly changed toward the anterior chamber. Splitting of the cornea in making the flap is not necessary. Accidents: perforation of the flap, falling of the trephined disk into the anterior chamber, hemorrhages, rarely loss of vitreous, nonprolapse of the iris. Iridectomy is to be desired as it gives better results than trephining without it. Once there was a rapid loss of vision after trephining. Eight late infections, loss of the eye in five. In simple glaucoma the daily dose of miotics which will keep tension down is determined; if there is warrant for believing that this treatment will be carried out it should be instituted, but if not the eye should be trephined. The operation should be done also if pilocarpine is not permanently of benefit, or if the field contracts. Contraindications are diseases of the lacrimal sac, conjunctivitis, fragile conjunctiva, suppurations of the skin. In inflammatory glaucoma also miotics should be resorted to first; if the increase of tension has not subsided within twenty-four hours an operative intervention is indicated. Iridectomy usually suffices, it is performed more quickly, is less painful, and the escape of the aqueous is more gradual. When the visual field is contracted an operation is less dangerous than expectant treatment. In absolute glaucoma intervention may be to relieve pain and to prevent degeneration. In secondary glaucoma one must weigh carefully whether any and what intervention is necessary. In buphthalmos trephining is no better than sclerotomy.

SEIDEL (98, **Permeability an essential property of the scleral scar**) concludes that the effect of trephining depends on the permeability of the scar, that in glaucomatous eyes the height of the tension and the quantity of aqueous which escapes in the time unit stand in causal relation to each other, and finally that the intraocular tension in chronic glaucoma depends upon an insufficient escape of aqueous.

GIFFORD (95, **Peripheral iridotomy (Curran) in the treatment of glaucoma**) has operated upon thirty eyes by the method advocated by Curran, who uses a knife needle with which he pierces the cornea and then the iris at its periphery thus

making a small hole about 1*mm* in size. He thinks that hypertension in cases where the anterior chamber is shallow, is due to the fact that the lens by pressing on the posterior surface of the iris interferes with the circulation. The hole in the iris acts in a way to establish the communication between the two chambers. Gifford has found some difficulty in carrying out the technic and had two cases which were not satisfactory but on the whole had favorable results. If the results prove to be permanent he is of the opinion that this operation, or some modification of it, may be destined to supplant to some degree those now in use.

ALLING.

HERTEL and CITRON (96, **Osmotic pressure of the blood in glaucomatous persons**) find the average osmotic pressure of the blood lower in persons suffering with glaucoma than in others. A pressure of over 6000 was found in 63% of healthy people, with glaucoma in only 16. Lower than 5600 was not met with at all with normal eye tension, while it was present in 45% of the glaucoma cases. Such differences of pressure may have an influence on the tension of the eye.

KOYANAGI (97, **Glaucoma and cataract after wasp sting**) was induced by a case of this nature to investigate the injuries produced in the eye by stings of wasps and bees. He finds that the poison induces a purulent conjunctivitis often leading to necrosis. Necrosis appears about the sting wound in the cornea, followed by accumulations of leucocytes. At first there is a contraction of the pupil, but this soon passes off and gives way to a marked dilatation, which is to be ascribed to necrotic changes in the iris, especially of the sphincteric portion, consisting partly of hyaline degeneration, partly of a transformation of the muscle into lumpy masses. In one case the endothelium of the cornea was proliferated, extended to the iris and later formed a strong adhesion of the periphery of the pupillary portion outward. It is thought that the increase of tension in the case of the boy who suffered this injury was not due to the apposition of the strongly contracted iris to the posterior surface of the cornea, but to a proliferation of the endothelium which extended over the anterior surface of the iris and joined the root of the iris to the cornea. Such an increase of tension was not observed in the animals experimented

on, for which Koyanagi suggests the explanation that the aqueous secured new exits through the cornea robbed of its endothelium. The resulting cataract is thought to depend chiefly upon the direct action of the poison which enters from the anterior chamber, yet possibly it depends also on the toxic action on the cornea. The iris is further depigmented through degeneration of the chromatophores and of the pigment in the posterior layer.

XIV—THE VITREOUS, RETINA, AND OPTIC TRACT.

99. EPPENSTEIN. **Primary tuberculosis of the retina.** *Archiv f. Ophthalmologie*, ciii., p. 154.

100. FEJER, J. **The treatment of tumors of the hypophysis.** *American Journal of Ophthalmology*, January, 1922.

101. FINNOFF, W. C. **Some impressions derived from the study of recurrent hemorrhages into the retina and vitreous of young persons.** *Transactions of the American Ophthalmological Society*, 1921.

102. GIFFORD, H. **Late traumatic detachment of the retina. Its prophylaxis and importance from a disability compensation standpoint.** *American Journal of Ophthalmology*, November, 1921.

103. HEINE. **Milk injections in albuminuric retinitis.** *Münch. med. Wochenschrift*, 1921, No. 43.

104. HIRSCH. **Air embolism in the central artery of the retina after washing out the antrum.** *Klin. Monatsbl. f. Augenheilkunde*, November, 1921.

105. HOLLOWAY, T. B. **Snowball vitreous opacities.** *American Journal of Ophthalmology*, February, 1922.

106. SCHMIDT. **Milk injections in albuminuric retinitis.** *Münch. med. Wochenschrift*, 1921, No. 48.

107. WILLIAMSON. **Two cases of thrombosis of the retinal vein, one showing a hole, the other a star at the macula.** *British Journal of Ophthalmology*, February, 1922.

108. YANO, F. **Atypical circinate retinitis.** *American Journal of Ophthalmology*, October, 1921.

HOLLOWAY (105, **Snowball vitreous opacities**) calls attention to certain vitreous opacities which differ decidedly from the cholesterin crystals of synchysis scintillans, a case of this sort has been examined pathologically and described by Verhoeff under the title "asteroid hyalitis." The author has added four cases to those already reported. Instead of appearing as glittering showers they are dead white or lardaceous in appearance showing a tendency to be globular and not flat. The movements in the vitreous are also different. He does not

agree with Verhoeff in ascribing this condition to intraocular angiosclerosis. It is possible that the condition may be due to hypercholesterolemia of suprarenal origin. These bodies should not be confused with the whitish deposits sometimes seen in a detached retina nor with the large round corpuscle-like vitreous opacities found in severe types of uveitis, nor with the margins of exudate.

ALLING.

HIRSCH (104, **Air embolism of the central artery of the retina after washing out the antrum**) describes a case of this nature. The papilla was quite white, the arteries appeared to be interrupted by brilliant white, bloodless bands. The phenomenon disappeared in a few minutes, and subsequent ophthalmoscopic examinations revealed nothing.

HEINE (103, **Milk injections in albuminuric retinitis**) reports that in two cases the vision was greatly improved, in one from $\frac{6}{60}$ and $\frac{6}{36}$ to $\frac{6}{8}$ and $\frac{6}{6}$, in the other from $\frac{6}{60}$ and $\frac{6}{12}$ to $\frac{6}{8}$ and $\frac{6}{6}$. In two the vision remained the same in spite of the retinal disease, and in two others the vision of one eye improved while that of the other failed.

SCHMIDT (106, **Milk injections in albuminuric retinitis**) also reported a case in which an albuminuric retinitis was considerably influenced in this way.

Aside from a scleritic focus and a very few infiltrations of lymphocytes in the choroid and ciliary body, EPPENSTEIN'S (99, **Primary tuberculosis of the retina**) case showed foci of lymphocytes and epithelioid cells in the retina. There were infiltrations in the perivascular lymph spaces of the papillary vessels, especially of the veins. At the blind end of the sheath of the optic nerve were foci with epithelioid and giant cells. In the vitreous was a shrunken mass supplied with newly formed vessels coming from the papilla, which had caused a detachment. The freshest foci lay proximally, so the tuberculosis seemed to threaten to extend to the meninges, and it is recommended that when such an eye is enucleated a large piece of the optic nerve should be resected at the same time.

The pathology of holes at the macula has been a subject of speculation for many years. The condition is usually connected with trauma, and in this connection various theories have been started. Menteith Ogilvie favored the theory of

Tearing by Contre Coup, and Fuchs suggests a mild traumatic retinitis causing a serious exudate, which ruptures the internal limiting membrane. Coats supported the theory of œdema as being the underlying cause, and also stated that holes had been observed in non-traumtic cases. WILLIAMSON (107, **Two cases of thrombosis of the retinal vein, one showing a hole, the other a star at the macula**) brings forward two cases, in both of which there was thrombosis of the retinal vein, with the formation, in one, of a hole at the macula, and in the other of a star at the macula. It is suggested that in this latter case a hole would eventually have appeared. Both eyes were excised.

In each case there was no suggestion of trauma, but in both there were evidences of past irido-cyclitis. Williamson points out, and the micro-photographs, with which the article is illustrated, clearly show, that in each case there is a sub-retinal œdema of the macula region. This sub-retinal œdema, Williamson thinks, is the primary cause of the hole formation, degenerative changes occurring subsequently in the retina.

As to the causation of the sub-retinal œdema, Williamson suggests that in these two cases there is a toxic influence,—shown by the presence of the irido-cyclitis—acting on the retinal veins, causing thrombosis, and acting on the delicate macular choroidal capillaries, causing degeneration of their walls and consequent œdema. This fluid then collects in the potential space between the rods and cones and the pigment epithelium.

FINNOFF (101, **Some impressions derived from the study of recurrent hemorrhages into the retina and vitreous of young persons**) from his observation of five cases and a study of literature concludes that tuberculosis especially of the veins is the common cause although syphilis, focal infection, and hemophilia may play a part. The first changes are at the periphery of the retina and retinitis proliferans follows in most cases. The detachments are due to traction and not to sub-retinal hemorrhages. The disease is much more frequent in men and the prognosis is poor.

ALLING.

GIFFORD (102, **Late traumatic detachment of retina. Its prophylaxis and importance from a disability compensation**

standpoint) states that most detachments, in eyes not predisposed, are traumatic and may occur years after the injury. He relates a case in which the eye was struck by a sling-shot and showed immediately a slight hemorrhage in the anterior chamber and a cloudy vitreous. The only evidence which remained later was a small posterior polar cataract and a few vitreous opacities. Four years afterward however, the patient complained of disturbance in the vision and a detachment in the lower outer quadrant was discovered. He thinks that cases of perforating wounds and contusions should be treated with rest, binocular bandage and salicylates or pilocarpine sweats and should be warned against strain or shock for a number of years following the injury. ALLING.

YANO'S (108, **Atypical circinate retinitis**) patient was a boy of thirteen presenting in each eye an oval zone of white exudation including the macula and extending about the nerve on the nasal side. Vision was lost in the right and one tenth in the left. No cause for the disease could be found. It is interesting because of the age of the patient and its extension to the nasal side of the papilla. ALLING.

FEJER (100, **The treatment of tumors of the hypophysis**) reports two cases of pituitary tumor which were treated with X-ray appropriately arranged for necessary penetration. In the first case three treatments were given each lasting about three hours and there was a slight improvement in the vision which had been reduced to about $\frac{5}{10}$. The second case had been totally blind but under similar treatment regained a vision of $\frac{5}{10}$ with enlargement of the fields.

XV.—ACCIDENTS, WOUNDS, FOREIGN BODIES, PARASITES.

109. BELLOWS, G. E. **Gunshot wounds of brain with visual field defects.** *American Journal of Ophthalmology*, December, 1921.

110. BLECKMANN. **Causes of total blindness in war.** *Diss. Marburg*, 1920.

111. LI, T. M. **Hole in the macular region of both eyes due to simultaneous injury.** *American Journal of Ophthalmology*, January, 1922.

112. PILLAT. **A case of cysticercus subretinalis.** *Wien. klin. Wochenschr.*, xlii., p. 925.

113. VOGT. **A case of siderosis bulbi examined with the slit lamp microscope.** *Klin. Monatsbl. f. Augenheilkunde*, February, 1921, p. 277.

BLECKMANN (110, **Causes of total blindness in war**) found among 114 blinded during the war that twenty-six had had both eyes shot out, or so badly injured that the remains of the eyes had to be removed by operation. In forty-one one eye had been lost and the other blinded by a direct lesion from in front, or from the side (in 15 cases), or indirectly through a shotwound through the orbit and temple (16 cases). In twenty cases there was practical blindness caused in nine by a direct lesion, in eleven by shotwound of orbit and temple, in one by sympathetic ophthalmia which set in ten and a half months after the injury; after enucleation of the second eye the first recovered with a vision of $\frac{1}{6}$. Eight had been blinded by shot wounds of the occipital region. Three presented homonymous hemianopsia, one bilateral central scotoma, one a paracentral scotoma, three changes in the optic nerve which indicated cranial complications. In one case of blinding by bilateral phthisis bulbi a metastatic ophthalmia was ascribed to a suppurating wound of the thigh. Only ten cases were not traumatic: 1, Cystic tumor in the left temporal lobe with choked disk; 2, bilateral high myopia and detachment of the retina; 3, intraocular cysticercus; 4, bilateral very severe iridocyclitis; 5, central choroiditis; 6, optic atrophy after meningitis; 7, optic atrophy after choked disk; 8, genuine optic atrophy; 9, optic atrophy in multiple sclerosis; 10, iritis and keratitis in Werlhof's disease.

Observations made during the war have increased our knowledge of the visual area in the posterior cortex and there is substantial agreement among the British, French, and German reporters. It seems well established that the upper and lower borders of the calcarine fissure correspond to the upper and lower halves of the retina of the same side; that the macula is represented in the extreme tip of the area perhaps extending a little onto the lateral surface; that the cortex from before backwards, represents the retina from the periphery inwards to the macula. BELLOWS (109, **Gunshot wounds of the brain with visual field defects**) cites three cases which illustrate some of these points. The reason why the macula is so frequently spared in hemianopsia is explained by the fact that the cortical area is supplied both by the posterior and middle cerebral arteries so that the occlusion of one alone would still leave the

region unaffected. Defects in the lower fields are much more common because the lower visual cortex is in close proximity to the cerebellum and the great sinuses and wounds in this region are much more likely to be fatal. He thinks that the left field, if there is any difference, is likely to be smaller than the right but offers no explanation.

ALLING.

I.—THE EYE IN ITS RELATIONS TO GENERAL DISEASE.

1. ARLT. **Encephalitis lethargica ambulatoria.** *Klin. Monatsbl. f. Augenheilkunde*, 1921, p. 928.

2. BALL, J. M. **Errors in ophthalmic literature.** *American Journal of Ophthalmology*, May, 1922.

3. BELL, G. H. **Retinitis proliferans from anæmia produced by Hodgkins' disease.** *Ibid.*, July, 1922.

4. BREGAZZI. **Epidemic encephalitis.** *Deutsche Zeitschr. f. Nervenheilk.*, LXXII, p. 15.

5. BRAM, I. **Exophthalmos in exophthalmic goiter.** *American Journal of Ophthalmology*, August, 1922.

6. CALHOUN, F. P. **Ocular manifestations in a case of hypophysial syphilis.** *Ibid.*, December, 1922.

7. COLLIN. **A rarely cured tumor of the brain.** *Klin. Monatsbl. f. Augenheilkunde*, 1921, p. 121.

8. FLECK. **Isolated reflex immobility of the pupils in a healthy adult as an indication of congenital syphilis.** *Zeitschr. f. die gesamte Neurologie u. Psychiatrie*, 1921, p. 34.

9. GUTMANN. **Connection between diseases of the eye and of the teeth.** *Deutsche med. Wochenschrift*, 1921, p. 565.

10. HORNIKER. **Ophthalmoscopic studies in war nephritis.** *Arch. f. Ophthalm., Fuchs' Festschrift*, p. 104.

11. KEY, B. W. **Hypophysial disease probably of syphilitic origin.** *American Journal of Ophthalmology*, December, 1922.

12. KRAEMER. **Keratomalacia in erythrodermia desquamativa.** *Wien. med. Wochenschrift*, 1921, p. 1063.

13. LENZ. **Pathological anatomy of encephalitis lethargica.** *Klin. Monatsbl. f. Augenheilkunde*, 1921, p. 929.

14. LIBBY, G. F. **Epidemic encephalitis from the standpoint of the ophthalmologist.** *American Journal of Ophthalmology*, October, 1922.

15. MASON, V. R. **Optic Neuritis in Serum Sickness.** *Journ. A. M. A.*, Jan., 1922.

16. MCGUIRE, H. H. **Observations in a case of lipæmia retinalis.** *Ibid.*, November, 1922.

17. PATON, LESLIE. **Tabes and optic atrophy.** *British Journal of Ophthalmology*, July, 1922.

18. PERLIA. **Acute retrobulbar neuritis after inhalation of benzol fumes.** *Klin. Monatsbl. f. Augenheilkunde*, 1921, p. 109.

19. PROCKSCH. **Bilateral keratitis and iridocyclitis in erythema multiforme exudativum.** *Wien. med. Wochenschrift*, 1921, p. 1076.

20. SATTLER. **A case of retrobulbar optic neuritis as an early symptom of Graves' disease.** *Wien. med. Wochenschrift*, 1921, p. 1084.

21. SIDLER-HUGUENIN. **Choked disk in tetany.** *Arch. f. Ophthalm.*, cvii, p. 1.

22. WAETZOLD. **Eye disturbances from optochin and their prevention.** *Therapie der Gegenwart*, lxii, p. 96.

23. WOLF. **Eye disease in xeroderma pigmentosum.** *Arch. f. Augenheilk.*, 1921, p. 168.

ARLT (1, **Encephalitis lethargica ambulatoria**) observed a case of this nature which was marked by a left hemianopsia that passed away completely after a few weeks.

LENZ (13, **Pathological anatomy of encephalitis lethargica**) found no changes in the cerebrum and only slight ones in the ganglia. In the caudex cerebri, on the contrary, he found infiltration of the sheaths of the vessels, multiplication of cells and neuronophagia, especially in the region of the oculomotor nucleus. No hemorrhages were found. His findings incline him to think that the brain disease comes by way of the lymphatics rather than by way of the blood.

BREGAZZI (4, **Epidemic encephalitis**) discusses the manifold symptoms of this disease and points out that none of them are of themselves diagnostic, while the entire picture formed by them becomes so. Lumbar pressure is usually increased in acute cases. Some of the cases which came under his observation had supranuclear disturbances, some involvement of the spinal cord. Anomalies of the tendon reflexes were frequent. In one case he noted in addition to pareses of the extrinsic muscles a bilateral central scotoma which passed away in the course of three months.

LIBBY (14, **Epidemic encephalitis from the standpoint of the ophthalmologist**), after rehearsing the symptoms of this disease, records the history of five cases which showed muscular paralysis and optic neuritis. He regards encephalitis as a disease of great importance and believes that eye symptoms are present in practically every case. He thinks it is possible to diagnose encephalitis by eye symptoms alone if syphilis is excluded.

Among the corrections which BALL (2, **Errors in ophthalmic literature**) has made are the following: Wallace an American, not Brücke, first describes the ciliary muscle. The sebaceous glands of the lid should be called after Zeis. Argyll Robertson should be written without the hyphen. The so-called Saemisch's incision was described and practised by Guthrie. Iridotasis was first employed by Guthrie not Critchett. Sclero-corneal trephining was first done by Argyll Robertson. The cautery in corneal ulcer should be attributed to Gayet. The honor of first using suction for cataract extraction should be given to Hulen, and hyperopia is less correct and expressive than hypermetropia.

The variability of the visual fields in tabes and the disproportion existing between the loss of visual acuity and the loss of visual field in the disease is noteworthy. In this paper PATON (17, **Tabes and optic atrophy**) attempts to find an explanation on the pathological side. As syphilis can attack the central nervous system in so many ways it is always difficult to decide as to which of the pathological manifestations are essentially those of tabes. The main question, which becomes apparent, is whether the change is a parenchymatous or interstitial one. As regards the site of the lesion the posterior columns, the posterior root ganglia and the posterior roots all have their advocates. Later work seems to support the theory that the radicular nerve of Nageotte—that portion of the root which is nearest the ganglion and where the dura and arachnoid sheaths have joined into one, but where there is still a definite subarachnoid space—is where the lesion is primarily situated. Regarding the cranial nerves, other than the optic nerve, the oculomotor nerves are most frequently attacked. The site of the lesion here has been held to be in the nuclei. This, if the case is not what might be expected as by comparison with the cord it is the afferent nerve which is the more vulnerable and the anterior root shows a greater resistance to the disease. The idea, suggested by Sherrington, is mentioned namely that the fleeting diplopias of early tabes may be due to disturbances in the afferent proprioceptive impulses from the oculomotor muscles and the muscles of the head and neck. Later authors favor the point where the nerve passes through the dura as the primary point of invasion.

Regarding optic atrophy, the older writers held that the primary seat of the disease was in the ganglion cells of the retina. Much evidence can be produced against this theory, however, for example, it has been shown that there are no changes to be found in the retina unless there are coincident changes in the optic nerve, and that after complete optic atrophy normal ganglion cells can still be found in the retina. Stargard's conclusion is that the simple optic atrophy of tabes and general paralysis is the result of a chronic inflammation commencing in the neighborhood of the chiasma. Richter finds the changes in the optic nerve in tabes and tabo-paresis identical; the granulation tissue no longer playing the predominant rôle as in other tabetic processes. It is an exudative process in which cells of hæmatogenous origin are predominant and in this respect it is more allied to that found in general paralysis. Mott speaks of the greater tendency of cases with early optic atrophy to pass into general paralysis but Richter thinks that the different reaction of the optic nerve can be explained by reason of its purely ectodermal origin, the supporting tissue of the optic nerve being glial in origin whereas in other nerves it is mesodermal. It can now definitely be said that the origin of the disease is due to the actual presence of spirochætes and the old hypothesis of a meta-luetic toxin can be abandoned. It is still an unsettled question as to whether the locally produced toxin acts by producing a parenchymatous degeneration or by acting on the connective tissue causes a consequent nerve degeneration. Paton favors the view that both these factors are at work. With regard to the clinical features, Paton points out that the loss of dark adaptation is one of the very earliest signs of tabes; he also notes the occasional development of colored vision. Regarding the visual fields, two main types may be found: (1) early loss of visual acuity with peripheral contraction of the fields; (2) areas of profound loss side by side with areas of full acuity. Paton suggests that the first type of field is produced when it is chiefly the parenchyma which is involved and the second type occurs when the lesion is chiefly of the interstitial tissue.

COLLIN (7, **A rarely cured tumor of the brain**) reports the successful removal of an endothelioma of the dura weighing 150 grams from the convexity of the right occipital lobe. The

patient had presented brain symptoms for at least four years, but the presence of exophthalmos prevented the diagnosis being made for a long time. Later such symptoms developed as a left homonymous hemianopsia, bilateral choked disk, staggering with a tendency to fall to the right, and from these, together with an exophthalmos of the right eye, the diagnosis was made. Although some of the patient's troubles persisted after the operation, the exophthalmos hemianopsia and choked disk disappeared.

CALHOUN (6, **Ocular manifestations in a case of hypophysial syphilis**) reports the case of a young woman twenty-four years old, who gave evidence of hypophysial disturbance through increased weight, polyuria polydipsia, bitemporal headaches, and scanty menstruation. She presented double choked disk, concentric contraction of the field of vision, and a central scotoma for colors, together with a history of a mild attack of iritis. A decompression operation failed to improve the symptoms, but examination of the ventricular fluid gave a Wassermann of plus 4 although the blood had been negative. Intensive antisyphilitic treatment was followed by complete recovery.

KEY'S (11, **Hypophysial disease probably of syphilitic origin**) patient was a young man aged 19 who complained of diplopia, loss of vision in one eye, headache and nausea. On examination a paresis of the left external rectus and choked disks were found. The field of vision in one eye had 10° concentric contraction and the blind spots were enlarged. Wassermann tests were negative and X-ray examination showed no abnormality of the brain. Later X-ray examinations, however, revealed an enlargement of the sella with some erosion and diagnosis of tumor of hypophysis was made. The patient soon began to develop signs of acromegaly. The choked disks subsided under intensive antisyphilitic treatment but the erosion of the sella seemed to progress. The question was as to whether there was syphilitic lesion, congenital or acquired, of the pituitary or a non-specific tumor or cyst. The choked disk, X-ray findings, negative evidence of syphilis would indicate the latter while a retino-choroiditis, external rectus paralysis, lethargy and tolerance of Hg. would point toward a specific cause.

FLECK (8, **Isolated reflex immobility of the pupils in a healthy adult as an indication of congenital syphilis**) reports a case of this nature. The mother was syphilitic. The patient had never an eruption on the skin as a child; encephalitis and acquired syphilis were excluded. His blood gave a negative Wassermann, but a positive Stern reaction, his cerebrospinal fluid a positive Wassermann. The last disappeared after a year of specific treatment. The immobility of the pupils is regarded by the writer as a cicatricial symptom.

BELL (3, **Retinitis proliferans from anæmia produced by Hodgkins' disease**) shows drawings of a marked and typical case of retinitis proliferans which appeared in an anæmic young man who was suffering from swelling of the glands of the neck and axilla and bronzing of the skin. The patient had diseased tonsils which were enucleated. Bell thinks that attention to diet and focal infections might clear up the ætiology of more of these obscure cases.

In GUTMANN'S (9, **Connection between diseases of the eye and of the teeth**) first case a sharply defined swelling of the lower transition fold appeared five months after resection of the apex of the root of an incisor. The cause was a gangrenous process in the alveolar septum and root of another incisor, and the conjunctival œdema subsided after this had been treated. A similar œdema had formerly existed on the other side during the treatment of a maxillary empyema, caused by periodontitis, until all signs of trouble in the antrum had cleared up. To explain these symptoms he recalls the connections existing between the veins of the alveolar region and the antrum with the facial and ophthalmic veins. The second case had a fistulous opening near the left inner canthus which did not connect with the lacrimal sac. After extraction of the two incisors on the same side there was a free escape of pus, while pressure on the mucous membrane caused an escape of pus both here and from the fistula. In a third case a periostitis of the lower margin of the orbit was ascribed to a gangrenous process in the root of a tooth with a gold crown. In his last case an obstinate eczematous conjunctivitis recovered quickly after the extraction of carious roots from the upper jaw.

During 1916–17, HORNIKER (10, **Ophthalmoscopic studies in war nephritis**) examined 608 soldiers, suffering from renal

disease, with the ophthalmoscope. The commonest symptom he observed on and about the optic nerve was œdema, diagnosed chiefly through the presence of pathological retinal reflexes and the behavior of the small vessels. He found hemorrhages in the retina in 11.5% of the cases, mainly in those having high blood pressure, as in retinitis albuminurica, which constituted 13% of the cases that came under observation. In some cases the retinitis was associated with choroiditic changes, and in one pigment striæ simultaneously with hemorrhages appeared very early, which rendered the explanation of the striæ as due to hæmatogenous pigmentation very improbable. The changes observed in the vessels varied; in some cases the vessels were normal; they were small in most cases of chronic diffuse glomerulonephritis, in all cases of malignant sclerosis, and in one case of uræmic eclampsia; very often, especially in acute diffuse glomerulonephritis, the little vessels about the macula were very tortuous. Although not present in all cases, an ischæmia of the retinal vessels can be seen, so that the writer believes that in all forms of nephritis the retinal vessels have to go through a spastic stage which can be recognized only occasionally because it passes away so quickly. Quite similar symptoms to those of nephritis may appear in other ischæmic diseases, but as ischæmia is accustomed to cause serious functional disturbances, which are not common in nephritis, Horniker thinks there must be a toxic agent which affects particularly the region involved in the ischæmia.

In McGuire's (16, **Observations in a case of lipæmia retinalis**) case the blood in the retinal vessels appeared to be a light salmon color while the choroidal vessels were filled with normal colored blood. The vessels were not enlarged or tortuous. The blood fat estimation was 8.6%. He has found no case not associated with diabetes. He is of the opinion that more cases would be discovered if the internist were to make use of the ophthalmoscope as a routine.

Bram (5, **Exophthalmos in exophthalmic goiter**) notes that exophthalmos or goiter are not essential in diagnosis of the disease. Exophthalmos rarely precedes but usually follows the other characteristic manifestations. It is probably the basis on which the other ocular signs depend. A goiter and

exophthalmos are not likely to occur separately. It is unusual for one eye affected to be alone. In the process of recovery the protrusion of the eyeball is usually the last sign to disappear.

SATTLER'S (20, **Retrobulbar neuritis an early symptom of Graves' disease**) patient developed a retrobulbar neuritis for which no explanation could be obtained from a general examination. He was advised not to use tobacco and given iodide of potash, but under this treatment the vision continued to deteriorate and the patient became emaciated. More than a year after the discovery of the central scotoma an exophthalmos appeared and directed attention to other symptoms of Graves' disease, tachycardia, Graefe's symptom, and a slight tremor of the fingers. The occurrence of retrobulbar neuritis in this disease is very rare, but in this case it appeared to be an early symptom.

KRAEMER (12, **Keratomalacia in erythrodermia desquamativa**) saw in an infant which had been suffering from erythrodermia desquamativa for thirteen weeks, croupous conjunctivitis and finally xerosis of the conjunctiva and keratomalacia. Neither cornea became perforated, and recovery took place leaving keratectasia in the left eye, but only a circumscribed macula in the right. Little is known of the fundamental disease, but it appears to be a disease of nutrition. The infant was the third child of healthy parents, nursed, and was continuously in good surroundings.

PROCKSCH (19, **Bilateral keratitis and iridocyclitis in erythema multiforme exudativum**) describes a case of this nature. Although the symptoms in the acute stage were fairly severe, there was a good recovery with full vision. It was thought that the keratitis was metastatic and not caused by the conjunctivitis which was present; the iritis resembled that met with in other infectious diseases with exanthemata.

WOLF (23, **Eye disease in xeroderma pigmentosum**) observed an involvement of the eye in a case of xeroderma pigmentosum in a girl 6 years old whose parents were nearly related. Numerous yellow brown spots covered all exposed parts of the body, but were most marked on the head. On the left cheek was a tumor as large as a cherry. In places the skin was atrophic and showed teleangiectasiæ, scales, and warts.

There was a slight ectropion of the lower lids with vascularization of their margins. The left eye presented a pannus-like condition, diffuse opacity and deposit of numerous roundish spots, together with an irregular mass of pigmented tissue as large as a bean upon the conjunctiva and cornea. The right eye presented a tumor of the cornea with an uneven surface and a wall-like definition, which left only a small part of the cornea clear. The tumor was removed and appeared microscopically to be probably a carcinoma. Wolf thinks that possibly the disease on the eye was analogous to that of the skin, and that the pannus and the other changes were the remains of a broken down tumor with subsequent cicatrization.

PERLIA'S (18, **Acute retrobulbar neuritis after inhalation of benzol fumes**) patient spent two hours cleaning an oil pump with benzol. Shortly afterwards he was taken sick with dizziness and vomiting, and after two days he could no longer see. A week after this occurrence there was a minimal reaction to light of each pupil, amaurosis of the left eye, while the vision of the right was reduced to counting fingers at 2½ meters. Nothing definitely pathological could be found with the ophthalmoscope. Vision finally improved to counting fingers at 6 meters with the right eye, at ¾ meter with the left. At this time the ophthalmoscope showed temporal atrophy of the right optic nerve, total atrophy of the left. The right visual field was contracted above and on each side, with loss of power to distinguish red and green, the left was only a paracentral trace.

WAETZOLD (22, **Eye disturbances from optochin**) says that a harmful quantity of this drug may be introduced not only by a single large, but also by repeated small doses, which may be retained as a result of functional disturbance of the liver and kidneys. Hypersensitiveness to optochin rarely exists. Adults should receive 0.25 gram not oftener than four times daily. Children much less.

SIDLER-HUGUENIN'S (21, **Choked disk in tetany**) patient was first operated on for goiter at the age of 27. Twelve years later the goiter had grown again and attacks of tetany had set in, and she was operated on again. Some days later there were marked symptoms of tetany and impairment of the pupillary reactions. Three months later a bilateral choked

disk was discovered, with subsequent opacity of the lens and symptoms of iridocyclitis. Then increase of tension called for an iridectomy in each eye. The cataract was removed from the right eye, but the eye had to be enucleated after eight months because of painful phthisis. Finally the patient died. The microscopic examination of the posterior segment of both eyes showed a swelling of the papilla, absence of all signs of inflammation, and normal conditions in the optic nerve. In the tissue of the papilla little hollow spaces existed between the fibers, but these reached only to the lamina cribrosa. It is therefore evident that choked disk may occur not only as a consequence of intracranial pressure, but also by various toxic influences acting upon the vessel walls.

The syndrome that follows the introduction of a foreign protein into the veins has been described as serum sickness. The symptoms are fever, malaise, urticaria, and glandular enlargement, sometimes albuminuria also.

MASON (15, **Optic neuritis in serum sickness**) has observed three patients who showed optic neuritis after injections of antipneumococcus serum. One died, but the others recovered completely. The evidence at hand seems to warrant the assumption that optic neuritis combined with increase in the cellular and globular content of the cerebrospinal fluid may occur as the result of the serum treatment.

(*To be continued.*)

BOOK REVIEWS.

VII.—**The Operative Treatment of Glaucoma.** By Colonel H. HERBERT, I.M.S. (retired), p. 152, W. Wood & Co., New York, 1923. Price $3.00 net.

Colonel Herbert was one of the first to realize the inefficiency of iridectomy in certain forms of glaucoma and has worked persistently for twenty years in the endeavor to repeat conditions which had developed unexpectedly after cataract and glaucoma operations. The author draws attention to the decline of trephining on account of the dangers of a conjunctival bleb. A better substitute is in the author's opinion the iris inclusion method. Deliberate iris inclusion has been objected to because of the evil repute earned by accidental iris prolapses. The author believes that more than one type of operation must be accepted in glaucoma. It is taken for granted that progress lies only along the line of subconjunctival drainage, avoiding late infection on one hand, hypotony and failure to relieve tension on the other. As the successful iridectomies in glaucoma showed uveal impaction and as iris prolapse after cataract operations, if subconjunctival, remained free from inflammation, subconjunctival iridencleisis in a wound of moderate size was taken up. If an iridectomy is successful, the cicatrix remains visible; conjunctival œdema is present, sometimes recognizable only by abnormal pitting on pressure and satisfactory relief of tension. A successful iridectomy is therefore an example of subconjunctival drainage. A leaking cicatrix causes diffuse or localized changes in the conjunctiva. When diffuse, it shows as simple œdema. In higher degrees of filtration œdema there is a milky opacity. Localized changes include the formation of clear vesicular spaces.

For practical purposes, chronic glaucomas fall into two groups—No. 1, readily reducible by miotics, No. 2, not reducible by drops. Group 1 is relieved by moderate drainage of linear iris-free filtering scars; there is no firm closure of the filtration angle. In group 2 there are firm adhesions of the surfaces at the filtration angle, and iris inclusion operations appear to promise well. The advantage of preparatory miotic treatment is self-evident.

Deliberate iris inclusion is objected to from a series of misapprehensions; it offers in the writer's judgment the safest as well as the most effectual means of dealing with the more severe and advanced glaucomas. A fully covered prolapse gives certainty of relief, with permanence, safety, and technical ease and with only one danger, early infective complications —including sympathetic ophthalmitis—if due precautions are not taken. To be effective, a partial lining of both surfaces of the wound must be present to prevent healing. Infection is prevented by thorough conjunctival asepsis. The author is convinced that advance in the treatment of severe chronic glaucoma lies in the application of the iris-inclusion method. The vesicular conjunctival patch with its late infective danger should become a thing of the past. If all fistulous cicatrices, clinically iris-free, are in reality iris-containing opposition to iris incarceration should fall. Trial of iris impaction is surely now imperative.

As for trephining, which the author himself has not practiced, it is rejected on account of the previously mentioned reasons.

The author recommends the two following methods: *Small-flap sclerotomy*, though its field of applicability requires precise definition. A conjunctival flap, *6mm*, is made at some distance from a *2mm* scleral incision, *2mm* from limbus. Lateral incisions in the scleral flap are then made by sawing movements of a blunt-ended, narrow knife, extending in the deeper layers of the cornea. Iridectomy is not done. Finger pressure in the after-treatment is important.

In buphthalmos the author advises a very limited anterior sclerotomy, when the sclera is very thin, and a small-flap sclerotomy in the earlier cases.

Iris inclusion operation is used for severe and advanced

glaucomas. Superficial fibrosis protects against infection and marginal filtration takes place through the stretched iris-loop. The end (transverse strip) of the iris prolapse is cut away, the iris is drawn well into each angle to be nipped there; the scleral wound should not measure more than 5*mm*, and the anterior chamber should be tapped peripherally. In the end result there is an elongated, opaque, gray swelling, with uveal tissue at each end, without any trace of conjunctival vesiculation. The author objects to Holth's meridional iridotomy for fear of leading to too localized drainage with vesiculation.

The author has given us a very instructive treatise, based on careful analysis of much practical experience. The book is filled with many observations of great value to those interested in glaucoma operations. The views on the behavior of the conjunctival flap after sclerotomy methods are particularly important. The methods advocated are carefully described, with reasons for the various steps and explanations for the resulting favorable action and avoidance of possible failure. A strong case is made for the iris inclusion method, and a general trial of this form of operation will undoubtedly be the result. There can be no doubt of certain unfavorable results following trephining and that progress must lie in the line of correcting these. Colonel Herbert has shown a way along these lines, for further study and investigation.

A. K.

VIII.—**Diseases of the Eye.** By Sir JOHN HERBERT PARSONS. IV edition, 660 pages, 21 plates, and 326 text figures. New York, Macmillan Co., 1923. Price $4.50 net.

Parsons' excellent text-book now appears in a new (the 4th) edition with a revised text and a number of important additions. These comprise a section on Preventive Ophthalmology, which treats of the causes and prevention of blindness and of the hygiene of vision. A study of the causes of blindness is an illuminating accessory to knowledge of the diseases of the eye. The factors which produce blindness have a different rate of incidence at different ages, thus blindness from ophthalmia neonatorum, syphilis, phlyctenular disease, measles, trachoma, myopia, glaucoma and industrial condi-

tions, by disease or accident, varies in frequency according to age. Under Hygiene the author considers errors of refraction, illumination, reading and writing, and handicrafts.

This is an unusually well-balanced text-book. The scientific part is excellent, and the practical part is just as well presented. The illustrations should be noted, as they are well done, particularly those in color. The book is not bulky and covers the whole ground of ophthalmology, giving the student a solid foundation in the fundamentals. This is the best of the medium-sized text-books. A. K.

IX.—**Arboreal Life and the Evolution of the Human Eye.** By E. TREACHER COLLINS, London, with colored frontispiece and 25 illustrations in the text; 108 pages. Lea & Febiger, Philadelphia and New York, 1922. Price $1.73.

This is an enlargement of the Bowman lecture given in 1922, which is published in its present form at the instigation and with the aid of Dr. W. Campbell Posey.

In the introduction Collins points out the quality of vision required by the different types of mammals. The herbivorous terrestrial mammals require a wide range of vision, well developed smell and hearing, to allow them to detect danger over a wide area and a capacity for seeing in a dim light. For the carnivorous animals, which track their prey, binocular vision is a necessity and the ability to see fairly well in a dim light. For the arboreal mammals acute stereoscopic vision and good color vision is a necessity while their arboreal refuge does away with the necessity of seeing well in a dim light. Man having descended from the trees has by his needs obtained increased precision of his visual organs.

Thus the visual requirements needed for different forms of environment have resulted in different forms of architecture of the visual organs.

Chapter two deals with the field of vision. In animals needing a large monocular field the eye is set well out from the head and in some the outer wall of the orbit may be incomplete. The enclosure of the bony ring of the orbit in primates steadies the movement of the eyes in the interest of binocular vision. A large cornea increases the size of the field of vision and the clearness of peripheral objects, thus in some rodents the cornea

comprises nearly half the surface of the eyeball, while in the primates the cornea reaches its smallest relative size. In the same way, and largely for the same objects, the lens decreases in size; thus in primates the cornea and lens are so constructed as to sacrifice the monocular field in the interest of acute central fixation.

In ungulata the increased lateral diameter of the cornea, the horizontally enlarged pupil, and the extension of the retina forward on the sides are in the interest of the extension of the monocular field. Animals requiring panoramic vision have their eyes set laterally in the head. In the hare the optical axis is eighty-five degrees from the middle line. The movement of the axis towards parallelism is in the interest of binocular vision. It is interesting to note that in the human embryo the optic vesicles, at first opposite to one another, gradually turn, and before birth become parallel. The recession of the snout no longer needed for purposes for which it was originally intended goes hand in hand with the position of the eyes.

Conjugate movements of the eyes are present in the carnivora while monkeys and man have a highly developed spot of central vision and highly developed powers of convergence and accommodation. Together with the increased central vision in arboreal life, which man has retained after coming down from the trees, goes the increased field of fixation; the ability to bring the fovea to bear on objects in a wide circumference has been accomplished by increased mobility of the eyeballs and of the head.

In primitive animals sight and hearing were poorly developed compared to the sense of smell. The least developed eyes are found in nocturnal animals who have only rods in their retinæ. The various aspects of rod and cone vision are then described according to environment. Cone vision is most highly developed in the diurnal rapacious birds. Supplementary means have been developed to fit an environment having a low degree of luminosity. Some deep sea fish have evolved their own illuminating apparatus. Some fish and animals have a reflecting membrane, the tapetum lucidum, more or less completely developed. Monkey and man have no such reflecting device. The former because he is safe in his arboreal

abode, while man became a cave dwelling animal and finally discovered fire. In mammals more or less sensitive central areas are met with but only in primates is the fovea well developed. It is also found in some reptiles and in practically all birds. The depth of the fovea may be regarded as the measure of the sharpness of vision. The normal fovea is especially deep in swift fliers and in birds of prey.

Increased capacity for accommodation and for convergence is intimately associated with foveal development and with the adoption of arboreal life. The accommodative power is greatest in man, slightly less in apes and very considerably less in all the lower orders. This is well shown in the development of the ciliary body. The rudimentary ciliary body has only longitudinal muscle fibers. Man's eyes at birth are comparable to those of terrestrial mammals in the natural state who have not adopted arboreal life. His lenses are nearly spherical and his vitreous chambers of comparatively little depth. Vision is adapted for long distances with little capacity for focussing near objects. By expansion of the globe, flattening of the lens, increase of the vitreous in depth, man's eyes gradually reach the condition most suitable for his environment. If vision becomes restricted to short distances then myopia develops as it will also in the lower mammals confined in small compartments.

A rudimentary color sense is present in some dogs but is weak and this is probably true for some of the other mammals. It is probable that the color sense in monkeys is the same as that in man. The perception of small colored objects is essential to the existence of arboreal animals which live mainly on the fruits. For the existence of carnivorous and herbivorous mammals it is by no means essential.

The lecture closes with a chapter on the protective mechanism of the eyeball. The structure of the orbit, retraction of the eyeball by the choanoid muscle, the nictitating membrane, the lacrimal section are taken up in turn. The grasping of food with hands saves the eyes from much of the risk incurred when the food is seized directly with the mouth, therefore various protective mechanisms have disappeared in the higher orders. Collins concludes that the changes in the visual organs of mammals produced by the adoption of arboreal life

increased both the range and accuracy of their powers of observation. Thus the way was opened for the evolution of the mental faculties which enabled man's ancestors on their descent from trees to assume a predominant position in the animal kingdom.

It is difficult to express adequately one's appreciation of this very scholarly piece of work, which should be carefully studied in the original. It is a subject which has evidently occupied the author's attention for many years, and it is written in a style so simple and clear that the professional as well as the lay reader will read it with interest and enjoyment. G. S. D.

X.—**War Blindness at St. Dunstan's.** By SIR ARNOLD LAWSON, London, 148 pages. Oxford Medical Publications, Henry Frowde and Hodder & Stoughton, London, 1923. Price $2.50.

St. Dunstan's was started in 1914 and this is a general review of the cases there between that date and May, 1920. Total number of traumatic cases comprise sixty-six officers, and twelve hundred and sixty-eight non-commissioned officers and men. There were four hundred and eighty-nine non-commissioned officers and men blinded from non-traumatic causes. Eighteen months after the Armistice the total number of those blinded was estimated at one thousand eight hundred and thirty-three and disease was responsible for twenty-six per cent. of these.

Part one of this book deals with cases of traumatic blindness. Notable absence of sympathetic disease is commented on. There were no cases observed after the original injury, though a few developed after operative procedures. Shell fragments and bullets are aseptic and wounds made by them healed quickly and without much reaction. Many shrivelling stumps of this nature left in, which caused no trouble. Cases with complications in the accessory sinuses caused endless trouble. Traumatic cases included a great many choroidal ruptures. There were seventy-two cases of through and through bullet wounds of the orbit. In many cases the optic nerve was severed. Through and through wounds caused seventeen per cent. of the blindness from trauma. Notes of many characteristic cases are given.

Fracture of the skull,—in the fracture of the skull causing blindness, in seventeen cases the occipital regions were affected. Various field changes are spoken of. Unfortunately it was impossible to work up this very interesting class of cases completely. Two cases showed the paracentral loss so well described by Lister and Holmes and various French writers, which is very disabling although the central vision may be normal. It is too bad that more exact descriptions of these field changes could not be given. No charts are published. There were twelve cases of skull fracture other than occipital, mostly smashing blows in the frontal region. Blindness was produced in a variety of ways, direct smashing of the eye, injury to orbital contents by fracture of the walls and hemorrhage, thirdly by concussion. There were sixteen cases of concussion blindness which is so well pictured in the various war atlases. One case, however, is of special interest. This man was knocked down by a shell explosion and both eyes destroyed. Except for this he bore no evidence of external or internal injury. Lawson believes this was due to concussion but the complete evidence is lacking. A very large number of interesting miscellaneous injuries are then presented.

Part two deals with non-traumatic blindness and opens with some general reflections. It is not advisable to accept for service anyone whose defective disease is due to old inflammation of the uveal tract. This is also true in the case of one defective eye, especially when the patient is the subject of syphilis, chronic gonorrhea or the rheumatic affections. All cases of retinitis pigmentosa and chorioretinitis should be excluded from service and for this purpose an ophthalmoscopic examination of every candidate should be made, if only for the purpose of saving disability pension to the State. Military life is particularly apt to cause a recrudescence of some existing disease.

Then follows a detailed consideration of the cases of non-traumatic blindness. Lawson takes the reasonable position in regard to disability pensions, that if a man loses his sight in the service it is not for him to prove that service was the cause but for the State to disprove it. The cases cited are such as those we meet in civil life and comprise a large variety of conditions and a considerable portion of the book is given over to them.

Part three contains a chapter on disability pension and one on re-education. Lawson points out the greater difficulty in educating those who have previously had sight than in educating a blind child. Up to forty, theoretically, all should be trainable; between forty and fifty a few; and after fifty only the exceptional person. The degree of re-education in the different groups must depend on the person's intellectuality and on his mental attitude. Those who lost their sight suddenly are more difficult to re-educate than where the vision disappeared slowly. It is much easier to re-educate in an institution where all are suffering from similar difficulties. The importance of physical exercise and sports cannot be overestimated.

The experiments and experiences at St. Dunstan's are most interestingly described by the writer and the book well repays reading. G. S. D.

XI.—**Glaucome et Hypotonie (Glaucoma and Hypotony).** By Professor FELIX LAGRANGE, Bordeaux. 432 pages. Bibliothèque d'Ophtalmologie: V. Morax. Published by O. Doin, 8 Place de l'Odéon. Paris, 1922.

This is a study of the operative treatment of glaucoma and of hypotony, based on the following anatomic and physiologic conceptions. The intraocular fluids pass from behind forward and are excreted at the spaces of Fontana; a closure of this excretion angle with a chronic conjunctival thickening of the cellular meshes at the limbus results in the hardening of the eye. A subconjunctival fistulation at the limbus reëstablishes the course of the intraocular fluids and normalizes the ocular tension. The nature of glaucoma explains why in most cases a fistulizing method is efficacious, while it only rarely gives no result at all.

The subject is studied under the following headings: (1) hypertension; (2) changes in the light sense and in the color sense; (3) changes in central and peripheric vision; (4) the anatomic changes of the glaucomatous eye and of the optic nerve in particular; (5) diagnosis and prognosis of glaucoma; (6) non-operative and operative treatment.

The glaucomatous eye owes its hypertension to exacerbations of hypersecretion whose cause is situated in the nervous

system and not in the eye. In the field changes, the study of scotomas is of the greatest importance, as they are the first changes. Lagrange thinks that with the ordinary perimeter, if the object be small enough (½ to ¼*mm*), the same changes can be elicited as with the Bjerrum screen, especially if the light intensity is reduced. The typical Bjerrum and the paracentral scotoma not connected with the blind spot are regarded as pathognomonic of glaucoma. Rarely these scotomas continue to grow even after the ocular tension has been satisfactorily reduced by operation. This Lagrange explains by Schnabel's cavernous atrophy, indicating a pathological state of the optic fibers. As for changes in the light and color sense, the study of the macular region is sufficient; the threshold value, the smallest difference, and the minimum of saturation are to be determined. The minimum differential (U) is first affected. The light sense (R) itself is not affected except in advanced glaucoma. Color sense is affected only when the optic nerve becomes atrophic.

The anatomic changes consist in sclerosed vessels in the retina and in the iris (the choroid is always normal), atrophy of the ciliary body, and the generally accepted changes in the iris angle. The iris, pushed forward by the swollen vitreous, obliterates the spaces of Fontana. The swelling of the vitreous is due to the distention of the vitreous trabeculæ with an overproduction of aqueous humor. If this is not relieved, union in the filtration angle takes place. The degenerative changes in the optic nerve consist in lacunæ, upon which Schnabel insists with reason, in addition to vascular changes at the level of the anterior and long posterior ciliary arteries. The general state of the patient with glaucoma can be divided into nervous, vascular, or both combined. The former are the neuropaths with unduly excitable sympathetic system and liable to trophic disturbances. Cases where the tension abruptly varies are serious, also where the anterior chamber is absent for a long time; in this Lagrange advises the Heine operation (cyclodialysis). Suppression of the accommodation is a disturbing sign, as it indicates compression of the ciliary muscle. The picture of the optic disk is of great significance. A pallor out of proportion to the excavation is ominous; notwithstanding suitable fistulization, degenerative and trophic

changes (lacunæ) occur in the optic nerve. Narrow retinal vessels portend degenerative optic nerve changes.

If the nasal field contraction is very marked and even approaches the fixation point, an iridectomy is dangerous (p. 76) though a satisfactory explanation for this is not known unless it is due to trophic influences. This danger Lagrange avoids by doing a sclerectomy alone. A central annular scotoma is a very serious symptom prognostically. Color sense is usually well preserved. Under general treatment, calcium chlorid 3 g. daily is taken internally for its action on vascular permeability. This with miotics, massage, general hygiene, and correcting glasses constitutes the general treatment.

As to pathogenesis, the theories of Donders, Knies, and Schnabel together explain the cause of glaucoma. The amount of liquid in an eye depends on the function of the gland of the aqueous humor, the pars ciliaris retinæ, a secretory neurosis, then hypoexcretion (soudure de Knies) and trophic disturbances follow. The last explain the excavation of the optic nerve head, which is shown by the following: The cup is deepest in chronic glaucoma with only moderate tension. The excavation in eyes with normal tension is due to the degeneration of the nerve of arterial origin. In eyes exposed to great pressure the anterior segment and not the optic nerve gives way.

Lagrange's definition of glaucoma is that it is a dystrophic eye with vascular and nervous degenerations, with hypertension from hypersecretion, followed and accompanied by hyposecretion.

Under operations, the extraocular and intraocular operations other than sclerectomy are then taken up. Sympathectomy does not give a lasting result. Iridectomy acts by producing filtration in the scleral wound for a variable length of time from incarceration of the stump of the iris. In acute glaucoma, iridectomy is excellent; its good effect is proportional to the hardness of the eye. In sclerecto-iridectomy, the two excisions —scleral and iris—have opposite indications; sclerectomy is useful in proportion to the softness of the eye; iridectomy, as has been stated, is the reverse. This means that when the eye is hard, iridectomy may suffice, while in lesser degrees more of the sclera must be resected. In chronic glaucoma with

constant hypertension a favorable result is obtained in 40%, while in chronic glaucoma with intermittent hypertension the good results are only 20%. Therefore, in all cases (p. 175) other than acute glaucoma, and especially in chronic glaucoma with intermittent hypertension, a fistulization must be practiced preferably by a sclerecto-iridectomy.

The scar after a sclerectomy is incomplete and permits passage of fluids by one or more very small fistulæ. The scleral resection must be placed near the cornea, in the region of the canal of Schlemm. A scar may be fistulizing even if fistulæ cannot be seen with the naked eye. Lagrange objects to the term scar, as that is just the condition sought to be avoided, and in place of filtering scar, or fistulous scar, he prefers the word fistulization. An experimental study of fistulization in sclerectomy in a dog is reported, followed by a detailed description of the author's operation (p. 208), the scleral resection being made either with curved scissors or punch forceps. The scleral incision need only be 4*mm* wide. The attempt is made to divide the tendon of the ciliary muscle, thus opening into the choroidal spaces. If the iris angle is closed, the knife cuts the iris. A button-hole or complete iridectomy is made. Modifications and substitute operations are then described, emphasizing the priority and superiority of the author's method. The various forms of scars which result from this operation depend on a variety of factors. A filtering cicatrix results from a combined sclerectomy and iridectomy, free from all uveal entanglements; a cystoid scar, on the other hand, always follows incarceration of the iris. This traction of the iris is dangerous in the author's opinion, and a healing must be obtained without involving the uveal tract in any way. Three well-illustrated varieties of scars result from the author's operation, according to the form of operation and the state of the eye: (1) Thinning of the sclera. This happens when only a part of the thickness of the scleral flap is resected. While this does not give the same filtration as the others, its effect is not negligible. (2) Subconjunctival fistula. Under the transparent conjunctiva an opening in the sclera is visible. Generally this belongs to glaucoma simplex with feeble hypertension. (3) Ampulliform œdematous swelling of the conjunctiva. This is usual, and results when the entire thickness

of sclera is resected in an eye with plus tension. This is the most satisfactory outcome, and Lagrange has never seen any evil results for the eye, which he ascribes because no iris is caught in the wound. The variety of the resulting scar therefore depends on the degree of hypertension. The size of the scleral resection should be inverse to the hardness of the eye. The scleral flap should be resected along the line of the filtration angle, placed entirely in the anterior chamber and invading neither the ciliary body nor the cornea. Lagrange lays stress on making an opening between the suprachoroidal spaces and the anterior chamber. This is accomplished by dividing the ciliary tendon and cutting exactly in front of the root of the iris. Sclerectomy alone is indicated when (1) the glaucoma has intermittent or slight hypertension; (2) iridectomy is dangerous in cases where the field defect approaches the fixation point; (3) patients with pronounced nervous characteristics, where operations on the iris produce reflex visual inhibitions; (4) hemorrhagic glaucoma. If the tension is up enough to fear prolapse of iris, an iridectomy should be added. Sclerectomy with button-hole iridectomy is indicated when a complete iridectomy is dangerous. It preserves an intact pupil, increases action of miotics, and prevents iris prolapse. Sclerectomy with complete iridectomy is the operation most generally practiced, and is the operation of choice in chronic glaucoma with constant hypertension. The statistics of 104 cases are given as reported to the London Congress in 1913. The seven failures were: 1 acute glaucoma, 2 gradual loss of vision (trophic optic nerve trouble), 1 rapid inhibition after sclerecto-iridectomy, 1 hemorrhages in retina, 1 recurring hemorrhages in anterior chamber, 1 failure from sclerectomy being too small. There was no infection, no loss of vitreous, no expulsive hemorrhage, no traumatic cataract.

The author objects to trephining, because a satisfactory scleral resection must not invade the clear cornea nor injure the ciliary body and be covered by a thick layer of conjunctiva.

Anatomically it is important to remember two zones in the sclera forming the anterior wall of the filtration angle,—a scleral and sclero-corneal portion. The first is the zone of filtration. The length of the subconjunctival sclera in relation to the anterior chamber is measured between a line drawn

through the summit of the iris angle and a line indicating the attachment of the conjunctiva. This measures 0.750*mm* above (superficial) and 0.65*mm* below (deep). The sclerocorneal zone measures 1*mm* above and 0.80*mm* below. Sclerectomy (p. 355) should not be circular but elongated, 2 to 3*mm* long and a little less than 1*mm* wide. The operative wound should be 4*mm* long, and made with a Graefe knife. It should "fouiller" the summit of Fontana's spaces, so as to cut the ciliary tendon. "The operation is good when one does not cut this tendon, it is perfect if it is cut."

The second part is devoted to **Hypotony.** This means a tension less than 16 (Schiötz). It has two principal causes,—(1) a lesion of the ciliary body; (2) a nervous change in the secretion of the aqueous humor. A subject of ophthalmomalacia is fatigued, debilitated, suffering from hyposympathicotonia or vagotonia, the ductless glands are underacting, and the gland of the aqueous humor is deficient. A moderate trauma, especially to the eye, produces hypotonia, probably from a disorder of the ciliary body diminishing the secretion of the aqueous. Hypotony is observed in high myopia and in detachment of the retina. Examination of the tension in cases of high myopia found it to be reduced in one third and in a much greater proportion in detachment. To raise the tension of an eye, it is necessary to close the filtration channels. This can be done by converting the conjunctiva at the limbus and over the filtration angle into dense scar tissue, which prevents the egress of fluids from the anterior chamber. This is accomplished in part by subconjunctival injections of mercury cyanide or strong salt solution, or by resecting the conjunctiva (peritomy) and cauterizing the region of Schlemm's canal by the galvano-cautery. This operation has been done by the author in 30 cases of high myopia and in detachment. After six months a definite increase in tension was obtained.

This lengthy review seemed necessary to bring out the views of this master on many of the problems of glaucoma. While some of these may not be shared by other investigators, they furnish at least a working hypothesis for further study. A great deal of interesting material has been collected by the author to prove his conception of glaucoma and to show that his operation is not only the first but the best operation for

chronic glaucoma. The excellence of this operation is universally admitted, but that it is difficult in many cases cannot be denied. A. K.

XII.—**Lehrbuch der Augenheilkunde.** By Professor PAUL RÖMER, Bonn. 4th edition; 500 pages, 306 text illustrations and 32 colored plates. Urban and Schwarzenberg, Berlin and Vienna. 1923.

Römer, in rewriting the 4th edition of his well-known and deservedly popular book, has reduced the text to 500 pages. While reduction in the size of a text-book is generally desirable, the ease of reading the text in this case has suffered, just as the personality of the writer has been lost to a great extent. Treatment, in particular, is much too brief; this important part is barely outlined and is not sufficient for the beginner. The description of the clinical conditions is excellent, and the subject matter is brought up-to-date by the addition of important recent advances in ophthalmology. An essential part of the book are the many illustrations of which there are 306 in the text and 32 colored plates. A. K.

INTERNATIONAL CONGRESS OF OPHTHALMOLOGY (1925).

Notice of Postponement.

The Committee of British Ophthalmologists appointed to organize an International Congress in 1925 finds, with regret, that it is unable to do so in accordance with the conditions under which the British invitation was accepted by the Washington Ophthalmological Congress in 1922. It will be remembered that at Washington it was decided that the next Congress should be strictly international and that German should be one of the official languages. The Committee has since been informed that the Société Française d'Ophtalmologie, the Société d'Ophtalmologie de Paris and the Société Belge d'Ophtalmologie have passed resolutions to the effect that they feel themselves unable to participate in a Congress if Germans are invited. The Committee is of opinion that to proceed with the Congress in these circumstances would tend to perpetuate a schism in the ranks of Ophthalmology and militate permanently against the progress of the Science which all desire to promote. The Committee has, therefore, reluctantly decided to postpone the Congress.